EXHIBIT B
TO YOUNG DECL.

Michael B. McTaggart
Arizona Bar No. 030178
michael.mctaggart@nortonrosefulbright.com
Brett J. Young (pro hac vice)
Texas Bar No. 24042203
brett.young@nortonrosefulbright.com
NORTON ROSE FULBRIGHT US LLP
1301 McKinney, Suite 5100
Houston, Texas 77010-3095
Telephone: (713) 651-5151
Facsimile: (713) 651-5246

COUNSEL FOR DEFENDANT

SUPERIOR COURT OF ARIZONA
MARICOPA COUNTY

MARY MAJOR, individually, and as the representative of THE ESTATE OF ELWYN WEBB; JOSHUA WEBB, Plaintiff, v. SFPP, L.P.; KINDER MORGAN ENERGY PARTNERS, L.P.; KINDER MORGAN G.P., INC.; KINDER MORGAN OPERATING L.P. "D"; CALJET II LLC; SHELL OIL COMPANY; EQUILON ENTERPRISES LLC; CONOCOPHILLIPS COMPANY; ATLANTIC RICHFIELD COMPANY D/B/A ARCO PRODUCTS COMPANY; CIRCLE K TERMINAL LLC; BP PRODUCT NORTH AMERICA; EXXON MOBIL CORPORATION D/B/A MOBIL OIL CORP.; CHEVRON U.S.A. INC.; PRO-PETROLEUM INC.; VALERO MARKETING AND SUPPLY COMPANY; JOHN DOES I-V; AND BLACK CORPORATIONS I-V, Defendants.	**No. CV2018-003217** **DEFENDANT CONOCOPHILLIPS COMPANY'S THIRD SUPPLEMENTAL RULE 26.1 DISCLOSURE STATEMENT** **(Including Expert Disclosures)** **(Assigned to the Honorable Margaret R. Mahoney)**

Pursuant to Rule 26.1 of the Arizona Rules of Civil Procedure and the Court's Scheduling Order, Defendant ConocoPhillips Company ("Defendant" or "ConocoPhillips") hereby submits its Third Supplemental Disclosure Statement (Including expert disclosures).

26.1(a)(6). The name and address of each person whom the disclosing party expects to call as an expert witness at trial, the subject matter on which the expert is expected to testify, the substance of the facts and opinions to which the expert is expected to testify, a summary of the grounds for each expert opinion, the expert's qualifications, and the custodian of any reports prepared by the expert.

Retained Experts

ConocoPhillips submits the following names and addresses of the persons whose expert opinion(s) and conclusion(s) they expect to offer into evidence at the time of trial. ConocoPhillips reserves the right to supplement or amend this section in accordance with Rule 26.1(d)(2), as the discovery period is not over.

Dr. John Whysner, MD, PhD, DABT
Washington Occupational Health Associates
1140 19th St. NW
Suite 700
Washington, D.C. 20036

Dr. Whysner is a physician and scientist with a PhD in biochemistry. He is a board-certified toxicologist (Diplomat of the American Board of Toxicology). He has been engaged professionally for over 40 years researching and practicing toxicology in the areas of occupational and environmental medicine. His qualifications and experience are outlined in his Curriculum Vitae included with his report, which is produced herewith. *See* Whysner Report, attached as Exhibit "A." Also set forth in the attached report are his opinions, the basis for his opinions, and the facts and data upon which he relies. *See id.*

Dr. Ethan A. Natelson, MD
Houston Methodist Hospital
6550 Fannin St.
Suite 1101
Houston, Texas 7730

Dr. Natelson is a board-certified hematologist in clinical practice at Houston Methodist Hospital. He has more than 48 years' experience in clinical hematology and has been a member of the American Society of Hematology since 1971. His qualifications and experience are outlined in his Curriculum Vitae included with his report, which is produced herewith. *See* Natelson Report, attached as Exhibit "B." Also set forth in the attached report are his opinions, the basis for his opinions, and the facts and data upon which he relies. *See id.*

John W. Spencer, CIH, CSP
Environmental Profiles, Inc.
8805 Columbia 100 Parkway
Suite 100
Columbia, Maryland 21045

Mr. Spencer is a certified industrial hygienist and certified safety professional. His field of expertise is occupational safety and health. He has been an industrial hygienist for more than 36 years. Formerly, Mr. Spencer was with the National Institute of Occupational Safety and Health and led a group of industrial hygienists conducting research for the National Occupational Exposure Survey. As an industrial hygienist for the United States Coast Guard, he conducted thousands of exposure assessments inclusive of a wide range of products and chemicals. Mr. Spencer has also served as the President of the Chesapeake Section of the American Industrial Hygiene Association (AIHA) and was a member of the national AIHA Product Health and Safety Committee and the Emergency Response Planning Committee. He has also authored the Health and Safety Audits Manual, published by Government Institutes, and the AIHA Hazard Communication Guide,

published by AIHA. The American Board of Industrial Hygiene certifies him as a safety professional. His qualifications and experience are outlined in his Curriculum Vitae included with his report, which is produced herewith. *See* Spencer Report, attached as Exhibit "C." Also set forth in the attached report are his opinions, the basis for his opinions, and the facts and data upon which he relies. *See id.*

Non-Retained Experts

In addition to the above-referenced retained experts, the following witnesses are not retained or specially employed to provide expert testimony by ConocoPhillips but nonetheless may provide expert testimony. As these individuals are not retained specifically to provide expert testimony, ConocoPhillips is not certain of the full spectrum of opinions these witnesses may ultimately provide or of the materials or other information that may form the bases for their opinions. ConocoPhillips provides the following disclosures in accordance with Rule 26.1(d)(3) of the Arizona Rules of Civil Procedure based on the limited information it does have and what it expects these individuals may testify to and have relied upon. ConocoPhillips reserves the right to supplement or amend this section in accordance with Rule 26.1(d)(2), as the discovery period is not over.

Tom Thompson, CIH
c/o Norton Rose Fulbright
1301 McKinney, Suite 5100
Houston, Texas 77010
(713) 651-5151

Mr. Thompson began his career with Unocal in 1989 as a regional industrial hygienist. He began with Tosco in a similar role in 1998. He may testify on matters within his area of expertise, if any, which may include but not be limited to: (1) industrial hygiene monitoring and sampling,

including sampling for benzene exposure, was conducted at appropriate, regular intervals that were based off of an evaluation of historical records documenting exposure levels at the facility; (2) industrial hygiene monitoring and sampling, including sampling for benzene exposure, was conducted on persons who would be expected to have the highest risks of exposure, if any; (3) he cannot recall a single instance where industrial hygiene monitoring at the terminal in question, located at S. 51st Ave., Phoenix, AZ 85043, came back with benzene levels anywhere close to the action level, and the norm was for such samples to be non-detect; and (4) the terminal at issue was exempt from OSHA's benzene standard due to the state of the art and well-functioning vapor recovery system they had in place. It is expected that his opinions, if any, will be based on his experience at the terminal and his experience and expertise as an industrial hygienist. He has not been compensated for any work in this case, and ConocoPhillips is not aware of any previous expert testimony that would be required to be disclosed.

Dennis Gilmore
Douglas Kloepfer
c/o Norton Rose Fulbright
1301 McKinney, Suite 5100
Houston, Texas 77010
(713) 651-5151

Messrs. Gilmore and Kloepfer served as managers of the terminal in question during the relevant time period. They may testify on matters within their area of expertise, if any, which may include but not be limited to: (1) the terminal in question had adequate, well-functioning, state of the art safety features and components to prevent exposure to gasoline and gasoline vapors; (2) these components and features were effective in preventing exposure to gasoline and gasoline vapors; (3) the terminal conducted regular and sufficient inspections and maintenance to keep these

components and features operating properly; (4) Coastal—Mr. Webb's employer—was responsible for training him on the hazards of the chemicals he was transporting for them; (5) their knowledge of industrial hygiene monitoring and sampling conducted at the terminal; (6) terminal operators spent more time on the racks than individual drivers, and therefore monitoring their exposure was appropriate to evaluate the exposure risk, or lack thereof, to drivers; (7) the results from exposure monitoring of operators indicated that there was minimal at best exposure risk to drivers at the terminals and that the terminal's safety features were effective; (8) the terminal's steps for loading/unloading trucks at the terminal minimized exposure risks for drivers; (9) the expected exposure risk, if any, from loading/unloading gasoline at the terminal, dome-outs, and spills; and (10) Coastal's contract obligations. Further information regarding their anticipated testimony may be found in their depositions. It is expected that their opinions, if any, will be based on their experience at the terminal and expertise as terminal managers. They have not been compensated for any work in this case, and ConocoPhillips is not aware of any previous expert testimony that would be required to be disclosed.

Barry Detlefsen, CSP
Coastal Transport Company, Inc.
c/o Howry Breen & Herman LLP
Sean E. Breen
1900 Pearl Street
Austin, Texas 78705

Mr. Detlefsen is the vice president of safety for Coastal. He has served in that role since approximately 2001. Prior to that role, he served as the director of safety for Coastal. for approximately three months. His deposition suggests that he may testify on matters within his expertise, including but not limited to (1) Coastal had the responsibility to train its drivers on the

hazards of the chemicals they were transporting; (2) the adequacy of training provided to Mr. Webb from Coastal; and (3) appropriate steps taken in inspecting and maintaining tanker trucks driven by Mr. Webb and in loading/unloading them. Further information regarding his anticipated testimony may be found in his deposition. ConocoPhillips is unaware of what materials he may have relied upon, what compensation he may have received, or whether he has prior expert testimony to be disclosed.

Robert Super
117 Lesoine Dr.
Henryville, PA 18332
570-629-9596

Mr. Super worked as a driver who worked with Mr. Webb from the early 1980's to 2002. His deposition suggests that he may testify on matters within his expertise, including but not limited to (1) training provided to drivers such as Mr. Webb and whether, by way of that training or otherwise, they were educated on the contents and hazards of the chemicals they were transporting; (2) appropriate steps taken in inspecting and maintaining tanker trucks driven by Mr. Webb and in loading/unloading them; (3) drivers' expectation of the risk of exposure to gasoline and/or gasoline vapors while loading their trucks versus unloading them offsite from the terminals; and (4) the practice and exposure risks of top-loading versus bottom-loading at the terminals and the timing of the switch to bottom-loading gasoline at the terminals. Further information regarding his anticipated testimony may be found in his deposition. ConocoPhillips is unaware of what materials he may have relied upon, what compensation he may have received, or whether he has prior expert testimony to be disclosed.

26.1(a)(8)-(9). Tangible evidence & relevant documents.

Carrier Access Agreement Between Tosco and Coastal	PSXMAJ00000001-03
Industrial Hygiene Monitoring Data from Phoenix Terminal	PSXMAJ00000004-06
Coastal Delivery Data	PSXMAJ00000007-31
Schedule of Terminal Acquired by Tosco from Union Oil Company of California	PSXMAJ00000032-33
Coastal Delivery Data	PSXMAJ00000034
Terminal Access Agreement between ConocoPhillips and Coastal	PSXMAJ00000035-39
2012 ConocoPhillips Consent to Assign	PSXMAJ00000040-41
Master Motor Carrier Services Agreement Between ConocoPhillips and Coastal	PSXMAJ00000042-68
Bills of Lading regarding Eugene (Gene) Martin	PSXMAJ00000069-111
Coastal Delivery Data	PSXMAJ00000112 PSXMAJ00000113 PSXMAJ00000114-188
Organizational Charts	PSXMAJ00000189-235
Hazard Communication Policy	PSXMAJ00000236-242

Respiratory Protection Standard	PSXMAJ00000243-266
Sample Forms	PSXMAJ00000267-272
Air Emission and Vapor Recovery Unit Reports	PSXMAJ00000273-334
Air Permitting, Air Emission, and Vapor Recovery Unit Reports	PSXMAJ00000335-590
Air Permitting Documents, Air Emission Reports, Storage Tank Inspection Reports, and Vapor Recovery Unit Maintenance and Inspection Reports	PSXMAJ00000591-1394
Phoenix Terminal Work Instructions	PSXMAJ00001395-1652
Tosco Spill Response Plan for Phoenix Terminal	PSXMAJ00001653-1905
Air Permitting, Air Emission, and Vapor Recovery Unit Reports	PSXMAJ00001906-2131
Air Permitting, Air Emission, and Vapor Recovery Unit Reports	PSXMAJ00002132-2360
Phoenix Terminal Environmental Manuals	PSXMAJ00002361-2422
Air Permitting, Air Emission, and Vapor Recovery Unit Reports	PSXMAJ00002423-2686
Phoenix Terminal Strategic Plan	PSXMAJ00002687-2807

Health, Environmental & Safety Manual	PSXMAJ00002808-3211
Material Safety Data Sheets found at Phoenix Terminal	PSXMAJ00003212-3491
Phoenix Terminal Emergency Response Plan	PSXMAJ00003492-4382
Monthly Load Rack Inspection Reports	PSXMAJ00004383-4431
Storage Tank Inspection Reports	PSXMAJ00004432-4465
Information Regarding Terminal Safety Policies and Driver Information	PSXMAJ00004466-4470
Vapor Monitoring Reports	PSXMAJ00004471-4493
Seal Inspection logs	PSXMAJ00004494-4580
Driver Domeout Lists	PSXMAJ00004581-4592
Quarterly Emergency Shutdown Check	PSXMAJ00004593
Operator Inspections	PSXMAJ00004594-4610
Vapor Recovery Unit Preventative Maintenance Checklist	PSXMAJ00004611-4622
Vapor Recovery Unit Documents	PSXMAJ00004623-4636

Air Permit and Vapor Recovery Unit Documents	PSXMAJ00004637-4647
Vapor Recovery Unit Operation	PSXMAJ00004648-4660
Phoenix Terminal Facility Inspection Reports	PSXMAJ00004661-4864
Driver Infractions	PSXMAJ00004865-4866
Driver Information	PSXMAJ00004867-4947
Air Permit Documents	PSXMAJ00004948-4978
Industrial Hygiene Monitoring Data	PSXMAJ00004979-5042
Load Rack and Driver Loading Card Agreement for Elwyn Webb and Other Drivers	PSXMAJ00005043-5197
Driver Information	PSXMAJ00005198-5236
Phoenix Terminal Canopy Project Information	PSXMAJ00005237-5365
Vapor Recovery Pressure Monthly Check	PSXMAJ00005366-5376
Vapor Recover Unit Preventative Maintenance Checklist	PSXMAJ00005377-5596
Phoenix Terminal Masterplan	PSXMAJ00005597-5743

Material Safety Data Sheets found at Phoenix Terminal	PSXMAJ00005744-5795
Vapor Recovery Unit Maintenance Logs and Documents	PSXMAJ00005796-5859
Driver Domeot and Overfill Reports	PSXMAJ00005860-6278
Phoenix Terminal Domeout/Loadrack Information	PSXMAJ00006279-6333
Phoenix Terminal Inspection Reports	PSXMAJ00006334-6411
Phoenix Terminal Information	PSXMAJ00006412-6546
Phoenix Terminal Permit Information	PSXMAJ00006547-6716
Phoenix Terminal Operating Documents	PSXMAJ00006717-6961
TOSCO Training Materials	PSXMAJ00007950-7963
Internal TOSCO memoranda	PSXMAJ00007964-7966
TOSCO Industrial Hygiene Management Plan	PSXMAJ00007967-7999
TOSCO Draft Benzene Manual	PSXMAJ00008000-8014
Various medical records pertaining to Plaintiffs	See all medical records produced by Valero Marketing and Supply Company with its Third

	Supplemental Disclosure Statement served July 22, 2019.

NORTON ROSE FULBRIGHT US LLP

By: */s/ Michael B. McTaggart*

Michael B. McTaggart
State Bar No. 030178
michael.mctaggart@nortonrosefulbright.com
Brett J. Young (*pro hac vice*)
State Bar No. 24042203
brett.young@nortonrosefulbright.com
devin.wagner@nortonrosefulbright.com
1301 McKinney, Suite 5100
Houston, Texas 77010-3095
Telephone: (713) 651-5151
Facsimile: (713) 651-5246

COUNSEL FOR DEFENDANT CONOCOPHILLIPS COMPANY

COPY of the foregoing delivered this 22nd day of July, 2019, to:

Scott I. Palumbo
Palumbo Wolfe & Palumbo P.C.
2800 N. Central Ave., Suite 1400
Phoenix, Arizona 85004
(602) 265-5777
(602) 265-7222 (fax)
pwsp@palumbowolfe.com

Keith E. Patton, Esq.
Patton Law, P.C.
500 Cooper Ave. NW, Suite 100
Albuquerque, NM 87102
(505) 910-4800
(505) 910-4382 (fax)
keith@pattonlaw.com
(*pro hac vice pending*)

ATTORNEYS FOR PLAINTIFFS

EXHIBIT A

Report Regarding Mr. Elwyn Webb

John Whysner MD, PhD, DABT

July 22, 2019

Summary of My Assignment and Opinions

I have been asked to consider whether it has been established on the basis of relevant and reliable methodology that Mr. Webb's alleged myelodysplastic syndrome (MDS) could have been caused by his alleged exposures to gasoline and other petroleum products identified by the Plaintiff when he was as a truck driver during 1985-2015. I have been asked whether it has been established on the basis of relevant and reliable methodology that the gasoline and other identified products are capable of causing MDS, and in particular the type of MDS alleged to be relevant to this lawsuit by Dr. Robert Harrison. Also, I have been asked whether it has been established on the basis of relevant and reliable methodology that the alleged benzene exposures for Mr. Webb could have caused his alleged MDS.

In my opinion, to a reasonable degree of medical and scientific probability, the answer to these questions is no. [1]

My Qualifications, Training and Experience

I, John Whysner, am a physician and a scientist holding a PhD in biochemistry. I am a board-certified toxicologist (Diplomat of the American Board of Toxicology), and I have been engaged professionally for over 40 years researching and practicing toxicology in the areas of occupational and environmental medicine.

I am employed by Washington Occupational Health Associates, Inc., which is a consulting firm and medical practice in occupational and

1. Dr. Ethan Natelson concluded in his report that Mr. Webb's disease would best be characterized as a myelodysplastic syndrome/myeloproliferative neoplasm (MDS/MPN) or an atypical MPN. It is my understanding that a pathology review has not been completed based on the timing of retrieving the original tissue specimens from medical providers.

environmental medicine. From 2003 until 2014, I was also Associate Clinical Professor of Environmental Health Sciences in the Mailman School of Public Health at Columbia University. I was responsible for and gave most of the lectures in the graduate-level course "Fundamentals of Toxicology," which includes teaching the scientific basis for understanding whether certain chemicals have been found to cause cancer or other diseases in humans and the methodology by which scientists make this determination. Until 2002, I was Head of the Toxicology and Risk Assessment Program and Chief of the Division of Pathology and Toxicology at the American Health Foundation in Valhalla, New York. The American Health Foundation was a non-profit research foundation.

I was in the undergraduate program at Johns Hopkins University from 1961-1964, majoring in biology. From 1960 to 1964, I was also a research associate in the Department of Pharmacology at the University of Southern California. I then attended the University of Southern California, School of Medicine in the combined MD-PhD program, and from which I received my MD, and a PhD in biochemistry, in 1970. I attended University of Southern California under a grant from the American Cancer Society. In 1971, I completed an internship in pediatric medicine through the Albert Einstein College of Medicine in New York. From 1971 to 1973, I was a post-doctoral fellow at the National Institute of Child Health and Human Development at the National Institutes of Health in Bethesda, Maryland, where I studied brain cancer. I was concurrently in the United States Public Health Service at the rank of Lieutenant Commander. In 1973 and 1974, I served as Director of Biomedical Research in the Special Action Office for Drug Abuse Prevention in the Executive Office of the President of the United States.

Over the past 40 years, I have studied the toxicological effects—including the carcinogenic effects—of chemicals. I have published articles in the medical literature and written chapters for books concerning the relevance of animal tests for predicting human cancer. In addition, I have conducted laboratory research on brain, liver, stomach, breast cancer and leukemia involving research in humans, experimental animals and genotoxicity test systems. Many of my research projects on chemical-induced cancers in rodents have focused on special mechanistic studies to find

whether and at what dose these rodent findings are relevant to human cancer. The results of my research have been published in the peer-reviewed scientific literature.

I have served as a consultant to the International Agency for Research on Cancer (IARC), which is part of the World Health Organization. The IARC, headquartered in Lyon, France, is one of the world's leading cancer research institutions. Its Monograph series and carcinogenicity evaluation program, employing the well-known Group 1-4 classification scheme, is used routinely by the scientific community. My work with the IARC has involved the evaluation of chemical agents to determine their potential for causing cancer in humans and the classification of those chemicals. I was twice Chairman of one of the four subgroups of the Working Groups that produces the IARC's Monographs. In this capacity I was involved in the Working Groups that met to determine whether experimental data on specific chemical exposures involving rodent cancer bioassays (cancer studies in rats and mice) and genotoxicity tests (studies of cellular DNA damage) are relevant to human cancer.

I have served on the Environmental Medicine Committee of the American College of Occupational and Environmental Medicine, the nation's largest medical society dedicated to promoting the health of workers through preventative medicine, clinical care, research and education. I developed a course for this organization on chemical risk assessment and my collaborator on this project was another doctor from the U.S. Environmental Protection Agency.

I have consulted for Federal agencies including the Office of the Assistant Secretary for Health of the Department of Health and Human Services, the Centers for Disease Control, the Environmental Protection Agency, the National Institute of Environmental Health Sciences, the Agency for Toxic Substances and Disease Registry, the Department of Transportation, and the National Institute of Standards.

I have authored numerous risk assessments of metals, petroleum products, and polychlorinated hydrocarbons, including polychlorinated biphenyls (PCBs), chlorinated solvents and other chemicals. I have also performed risk assessments of emissions from power plants that have included the criteria air pollutants such as

particulates, sulfur dioxide, nitrogen oxides, ozone, carbon monoxide and lead. Also, I have provided testimony at public meetings and have been qualified as an expert in administrative hearings regarding trace emissions including dioxins from these facilities.

I have authored many articles that are published in peer-reviewed journals in the areas of carcinogenicity, tumor promotion, cancer mechanism and DNA changes associated with chemical exposure, among other topics. I am the author of a comprehensive review of mechanisms of benzene-induced AML, especially the possible role of genotoxicity and DNA reactivity (Whysner et al., 2000, 2004). I have published review articles on the toxicological effects and cancer mechanisms of many chemicals that cause cancer in animals and humans, and I have served as guest editor for a book on occupational cancer for the *Clinics in Occupational and Environmental Medicine* series. A copy of my curriculum vitae is attached to this report.

My Literature Search and Analysis

I have drawn on my experience as a toxicologist, researcher and physician. However, in order to form my opinions, it was necessary for me to examine Mr. Webb's alleged exposures to the products named by the Plaintiff in this case and the state of knowledge regarding health effects from benzene or gasoline. Mr. Webb never had AML but because it is alleged that his MDS (if in fact that was his disease) was cause by occupational exposure to benzene, and because the literature indicates that some cases of MDS can **become** acute myeloid leukemia (AML), and that some of the AML cases associated with high cumulative benzene exposures have been **preceded** by some forms of MDS, I have also reviewed the literature concerning benzene and AML. In order to do so, it was necessary for me to canvass the world's published scientific epidemiological and toxicology literature. My method for doing so breaks into eight parts, as follows:

- First, I reviewed product information including MSDSs produced by the Defendants to determine the general nature of the petroleum products

loaded (and unloaded) by Mr. Webb. I understand that a MSDS is not an analytical chemistry document, but it does provide general information on a product (e.g., unleaded gasoline).

- Second, I performed a literature search to determine whether and in what circumstances benzene or gasoline or other alleged petroleum products have ever been found to cause or be associated with MDS or AML.
- Third, where benzene, gasoline or other alleged petroleum products had been reviewed by some or all of the authoritative bodies described below for cancer in general and for MDS or AML in particular, I reviewed those analyses and evaluated independently the literature cited in these reviews including reports from the U.S. EPA, the IARC, the ACGIH, the ATSDR and the NTP.
- Fourth, I updated the authoritative bodies' analyses by performing a literature search of the relevant toxicology and medical and databases, especially regarding the dose of benzene required to increase the risk of MDS or AML.
- Fifth, I searched the information provided in Mr. Webb's medical records to determine the nature, history, and pathology of his specific illness. It appears that the disease diagnosis is different based on different medical records and Dr. Ethan Natelson has provided an analysis of this issue.
- Sixth, I reviewed the depositions of Barry Detlefsen, Mary Major and Joshua Webb.
- Seven, I reviewed the reports of Mr. John Spencer, CIH and Rachael M. Jones, PhD, CIH to understand the extent of Mr. Webb's alleged benzene exposures.
- Eighth, I reviewed the report of Dr. Robert Harrison.

I then prepared the following summary of my research on the question of whether gasoline and other petroleum products identified in this case are capable of causing MDS or AML. This research includes epidemiological findings, animal data, genotoxicity data, and the conclusions of authoritative bodies. This report provides a summary of my analysis of the available information and my conclusions about the ability of benzene or gasoline to cause MDS or AML, and the ability of alleged exposure to benzene in the named products to contribute to the development of Mr. Webb's alleged MDS. All of my opinions in this case are within a reasonable degree of medical/scientific certainty.

I reserve the right to amend my report should additional medical records be provided or should Dr. Harrison alter or amend his diagnosis of Mr. Webb's "*Myelodyplastic syndrome MDS/RAEB-1.*" I see no record of Dr. Harrison having conducted either a clinical encounter with Mr. Webb or an independent review of the medical evidence in this case (e.g., pathology review).

The Myelodysplastic Syndromes, Myeloproliferative Disorders, and Acute Myeloid Leukemia

Before turning to my review of the scientific literature concerning benzene and gasoline, it is instructive to understand the difference between MDS, myeloproliferative disorders and AML and to understand the different types of MDS. The formation of blood cells by the bone marrow is under homeostatic controls, which regulate the number of blood cells in the peripheral circulation (blood vessels). Blood cells have to be continually produced by the bone marrow because they normally have a limited life. The red blood cell lives about 120 days, so every four months the bone marrow must replace all of the red cells in the body. White blood cells are shorter lived and must be replaced every few weeks. Under normal conditions the bone marrow produces more blood cells as they are needed and senses when there are enough cells so that they are not overproduced. If more blood cells are needed, the homeostatic controls are released and more cells are produced. For example, when a person gives blood, the bone marrow will increase cell division among the

precursor cells to the blood cells, and when enough cells are produced, decrease production back to its original rate. On the other hand, if the bone marrow is damaged, it cannot replenish the blood cells needed, and the numbers of cells in the blood will decrease.

There are several myeloproliferative diseases whereby the above feedback control system is not properly operative. One common feature of these conditions is that the bone marrow overproduces blood cells, and this results in more blood cells in the peripheral circulation than normal. "Proliferation" occurs because the precursors of the blood cells are not responding to normal controls of cell division but, instead, are permanently turned on to reproduce. The causes of this failure of controls resulting in increased production of blood cells are not entirely understood but seem to involve acquired genetic changes that turn on cell division. This condition results in the bone marrow being hyperplastic, i.e., having more cells that normal. The most common of these myeloproliferative diseases is chronic myeloid leukemia (CML), affecting people from all walks of life. This disease has characteristic alterations in two of its chromosomes caused by the exchange of a part of chromosomes 9 and 22 resulting in a striking altered appearance of chromosome 22, called the "Philadelphia chromosome."

Acute myeloid leukemia (AML), also known as acute myelogenous leukemia, is a cancer of the myeloid line of white blood cells, characterized by the rapid proliferation of abnormal cells which accumulate in the bone marrow and interfere with the production of normal, healthy blood cells. Rather than point mutations in DNA being associated with AML, various translocations and deletions of chromosomes have been found. AML is the most common acute leukemia affecting adults, and its incidence increases with age. AML accounts for approximately 1.2% of new cancer cases and 1.8% of cancer deaths in the United States.2 AML as defined by the World Health Organization (WHO, 2017) encompasses acute erythroid leukemias (erythroleukemia), acute myelomonocytic leukemia and other acute leukemias of the myelocytic lineage. Some epidemiologists have used the

2 https://seer.cancer.gov/statfacts/html/amyl.html

terminology of acute nonlymphocytic leukemia (ANLL) instead of AML. In this report, I will use the WHO terminology for AML except where some studies have specifically used other terms. The cause of most cases of AML is unknown, but it has been causally associated with hereditary factors, exposures to ionizing radiation, cigarette smoking, obesity, certain chemotherapeutic agents, and sufficient exposures to refined benzene.

The myelodysplastic syndromes (MDS) are a diverse collection of conditions united by ineffective production of blood cells and varying risks of transformation to AML. In MDS, the bone marrow is unable to produce enough cells for the blood. Therefore, patients with this syndrome typically have anemia and/or abnormally low white blood counts and/or decreases in platelets. The most common forms of MDS that have been historically identified according to the French-American-British (FAB) system and studied are "refractory anemia" (RA), "refractory anemia with excess blasts" (RAEB) and "refractory anemia with ringed sideroblasts" (RARS), and "refractory cytopenia with multilineage dysplasia" (RCMD).

Several changes were made in the identification of the types of MDS when the World Health Organization developed its classification system that superseded the prior FAB system. RAEB is subdivided into RAEB1 and RAEB2, which have very different prognoses in terms of transformation to AML. Another FAB type RAEBT is now included within AML and RAEB2. RCMD was originally included in the RA category in the FAB classification scheme but now is considered by the WHO to be in its own category within MDS (WHO, 2017).

In the WHO classification published in 2008 (Brunning et al., 2008; WHO, 2008), RA was included within a more general classification refractory cytopenias with unilineage dysplasia (RCUD). Finally, there is a category of MDS with minimal dysplastic changes in the bone marrow, which is termed "unclassifiable" (MDS-u). It is not clear how MDS-u would have been classified in the FAB system assuming that such cases would have been diagnosed as MDS. Studies have found that a unique form of MDS with dysplastic eosinophils (MDS-Eo) is associated with high dose benzene exposure (Irons et al., 2005; 2010; Ruiz et al.,

1994). There is no indication in Mr. Webb's medical records that he had this type of MDS or was diagnosed with any such disease.

The WHO also established myelodysplastic syndrome/myeloproliferative neoplasm (MDS/MPN), which denotes conditions that have characteristics of both MDS and MPD. One type of MDS/MPN is chronic myelomonocytic leukemia (CMML), which was previously categorized with MDS under the FAB system. It can have characteristics of MPN and is currently considered by the WHO to be placed in category of MDS/MPN. Other types of MPN include atypical CML and MDS/MPN-U, which is the diagnosis suggested by Dr. Natelson based on his review of the medical evidence and his clinical expertise in diagnosis and treating hematological disorders.

Gasoline and Other Petroleum Products Have Not Been Shown to Cause MDS or AML, but Refined Benzene at a Sufficient Dose Can Cause MDS and AML

I have searched the world's medical and scientific literature to investigate the question of whether environmental exposures to gasoline or other petroleum products such as those alleged in this case have been causally associated with the development of MDS or AML. I found that there is no collection of information in humans or animals that has been reported whereby one could conclude that exposures to gasoline or these other products have caused either MDS or AML. Refined benzene exposure at a sufficient dose has been found to cause AML in humans and certain forms of MDS. There are demonstrated AML cases that have been preceded by the development of MDS.

Characterization of Products at Issue in This Case

Mr. Webb was a tanker trucker driver for many years. It is alleged that he was exposed to benzene from gasoline (identified in the complaint). Dr. Jones, the expert retained by Plaintiff, describes exposures to other petroleum products including avgas, diesel, jet fuel and ethanol.

Authoritative Bodies Have Addressed the Question of Whether Benzene or Gasoline or Other Petroleum Products Cause Cancer Including AML or MDS

The issue of whether chemicals can cause cancer has long been the subject of careful scientific inquiry by various organizations. Moreover, those organizations employ generally accepted scientific methods to determine whether a chemical causes disease including cancer. The carcinogenicity classification schemes adopted by these organizations determine whether or not the chemical is considered a human carcinogen, an animal carcinogen, or both. These schemes generally are confirmatory of one another and generally adhere to the same coherent set of rules. The principal bodies that are dedicated to studying this very issue and have reviewed benzene or gasoline are the following:

- U.S. Environmental Protection Agency (EPA)
- International Agency for Research on Cancer (IARC)
- National Toxicology Program (NTP)
- American Conference on Governmental Hygienists (ACGIH)
- Agency for Toxic Substances and Disease Registry (ATSDR)

As part of my work in this case, I have collected the documents produced by these organizations for benzene, gasoline, and other petroleum products. All of the organizations agree that gasoline and other petroleum products are not known to be human carcinogens. However, they agree that exposure to refined benzene at a sufficient dose has been found to cause AML.

The fact that these organizations recognize the difference between gasoline and benzene (toxicologically) is highly significant.

Conclusions of Authoritative Bodies Regarding the Carcinogenicity of Benzene. Gasoline and Other Petroleum Products.

USEPA has not designated any gasoline or other petroleum products in Group A (Carcinogenic to Humans); IARC has not listed any in Group 1 (Carcinogenic to Humans); NTP has not designated any in Group K (Known to be a Human Carcinogen); and ACGIH has not designated any in Group A1 (Confirmed Human Carcinogen).

In all cases, these organizations have evaluated benzene separately from gasoline and other petroleum products and have classified gasoline and other petroleum products in lesser designations so that they cannot be said to cause cancer in humans. That is highly significant.

These organizations acknowledge that there is published data showing that they may cause tumors in experimental animals, but insufficient or limited information exists to establish them as causing cancer in humans. Refined benzene has been designated by the IARC, NTP, EPA and ACGIH to be a known human carcinogen. However, as will be described below, products containing benzene are reviewed separately by these agencies and are not assumed to be human carcinogens simply because they contain small amounts of benzene.

The International Agency for Research on Cancer evaluated the evidence for benzene in its seventh monograph. The IARC (1974) concluded that a relationship between leukemia and benzene exposure was suggested based primarily upon case reports and a case-control study in Japan. However, this later study could not rule out other exposures as the causes of leukemia. The IARC also found that benzene had been tested only in mice by subcutaneous injection and skin application. The data reported did not permit the conclusion that carcinogenic activity had been demonstrated in animals. The incidences of leukemia in some of these tests were no different from controls.

In 1974, the NIOSH addressed the issue of whether benzene causes leukemia (NIOSH, 1974) and said the following: "Whether the alterations in marrow function observed from benzene exposure actually induce malignant changes is not

conclusive; nevertheless, the possibility that benzene can induce leukemia cannot be dismissed." Therefore, it is clear that the NIOSH in 1974 had not concluded that benzene caused leukemia. Also, the ACGIH in 1975 proposed that benzene be designated in category A2 as a suspected but not a known human carcinogen.

NIOSH recommended that the Occupational Exposure Standard for benzene should be decreased in 1976. This proposed standard was based upon its conclusion that benzene was a leukemogen in humans (NIOSH, 1976). The U.S. EPA found that benzene was a human carcinogen in 1979 (U.S EPA, 2019). The National Toxicology Program (NTP, 2016) listed benzene as a human carcinogen in 1980 due to "increased mortality from leukemia (mainly acute myelogenous leukemia)." And in 1982, the IARC (1982) re-evaluated the information on benzene and concluded the following:

> "It is established that human exposure to commercial benzene or benzene-containing mixtures can cause damage to the haematopoietic system, including pancytopenia. The relationship between benzene exposure and the development of acute myelogenous leukaemia has been established in epidemiological studies."

In 1990, the ACGIH concluded that benzene was a confirmed human carcinogen and placed in category A1.

Therefore, benzene has now been designated by the IARC, NTP, EPA and ACGIH as a known human carcinogen based on its ability to cause AML (IARC, 1982; 2012; U.S.EPA, 2014; NTP, 2016; ACGIH, 2001). Recently, the IARC has affirmed their cancer classification of benzene (IARC, 2018). As will be described below, petroleum-based products are not classified as human carcinogens by these very same organizations and agencies even though they contain small amounts of benzene.

The petroleum product, gasoline, has been reviewed in depth by several of these authoritative bodies. Gasoline has historically contained 1-5% benzene and during a portion of Mr. Webb's career may have contained 1-3% benzene (IARC, 1989a). None of the authoritative bodies have found that gasoline is a human carcinogen in general or that it has been shown to cause AML. The IARC evaluated gasoline (automobile and avgas) and found inadequate evidence in humans and only

limited evidence of carcinogenicity in experimental animals (IARC, 1989a). Therefore, the IARC classified gasoline in Group 2B – "possibly carcinogenic to humans" - even though it acknowledged that gasoline contains benzene. The 2B classification was based on animal data.

The ACGIH has classified gasoline in Group A3, which is an animal carcinogen of unknown relevance to humans (ACGIH, 2014). The NTP has not listed gasoline as a carcinogen (http://ntp-server.niehs.nih.gov/), and the EPA has not classified gasoline as a carcinogen (www.epa.gov/IRIS). The ATSDR (1995) has stated the following:

> "....gasoline has only been shown to cause increased incidences of renal cell tumors in male rats (a finding that is not considered relevant to humans) and liver tumors in female mice. Therefore, there is no conclusive evidence to support or refute the carcinogenic potential of gasoline in humans or animals based on the carcinogenicity of one of its components, benzene."

According to the ATSDR (1995), American gasoline typically contained 0.5-2.5% benzene.

Components of gasoline, such as ethylbenzene, toluene and xylene, have also been reviewed by these authoritative bodies, and none has been found to cause cancer in humans even though as commercial products they also contain benzene as a contaminant. Ethylbenzene is classified as Group D by the EPA. The IARC (2000) found that ethylbenzene is considered to be an animal carcinogen but not a human carcinogen (Group 2B). The ACGIH has classified ethylbenzene in Group A3, which denotes an animal carcinogen of unknown relevance to humans (ACGIH, 2014). The NTP has not listed ethylbenzene as either a human or animal carcinogen. The IARC has evaluated toluene and classified it in Group 3 based upon insufficient data for carcinogenicity in humans and in animals (IARC, 1999a). Likewise, the EPA has placed toluene in Group D (www.epa.gov/IRIS) and the ACGIH in Group A4 based on the same criteria (ACGIH, 2014). The IARC has evaluated xylene and classified it in Group 3 based upon insufficient data for carcinogenicity in humans and in animals (IARC, 1999b). Likewise, the EPA has placed xylene in Group D

(www.epa.gov/IRIS) and the ACGIH in Group A4 based on the same criteria (ACGIH, 2014).

The IARC reviewed jet fuels, which are primarily composed of kerosene, and concluded that there was inadequate evidence for cancer in experimental animals and in humans. Therefore, they placed jet fuels in Group 3, which is the lowest category that IARC routinely uses in their classification system (IARC, 1989c). The ACGIH has placed kerosene in Group A3, which denotes a finding of cancer in experimental animals of unknown relevance to humans (ACGIH, 2014). As stated by the ATSDR (1998, p. 67) in the monograph on JP-5 and JP-8, "The available epidemiological studies are generally inconclusive, since they cannot reliably associate exposures to jet fuels with the adverse effects reported."

Another petroleum product, diesel fuel, has been also reviewed in depth by some of these authoritative bodies. None of the authoritative bodies have found that diesel fuel is a human carcinogen in general or that it has been shown to cause AML. The IARC evaluated diesel fuel and found inadequate evidence in humans and only limited evidence of carcinogenicity in experimental animals (IARC, 1989d). Therefore, they classified marine diesel fuel in Group 2B – possibly carcinogenic to humans, but light diesel fuels in a lesser designation Group C. The ACGIH has classified diesel fuel in Group A3, which is an animal carcinogen of unknown relevance to humans (ACGIH, 2014). The NTP has not listed diesel fuel as a carcinogen (http://ntp-server.niehs.nih.gov/), and the EPA has not classified diesel fuel as a carcinogen (www.epa.gov/IRIS).

IARC evaluated the carcinogenicity of gasoline and diesel engine exhaust (IARC, 2014). In its overall evaluation, IARC stated the following:

> "There is sufficient evidence in humans for the carcinogenicity of diesel engine exhaust. Diesel engine exhaust causes cancer of the lung. A positive association has been observed between exposure to diesel engine exhaust and cancer of the urinary bladder. There is inadequate evidence in humans for the carcinogenicity of gasoline engine exhaust."

Consequently, IARC did not find that either gasoline or diesel exhaust was capable of causing AML.

Summary of the Medical Literature Concerning Whether Petroleum Products Including Benzene Cause Acute Myeloid Leukemia

I have employed a generally accepted scientific methodology for assessing causation, the elements of which were described by Sir Austin Bradford Hill (Hill, 1965). Many of these elements are also included in the Surgeon General's report on smoking and health (U.S. Public Health Service, 1964), which serves as a precedent for determination of causality (e.g., U.S. EPA, 2005). This methodology is applied to the results of relevant, well-conducted human epidemiology studies. These elements address the question whether observed statistical associations between a chemical exposure and a specific disease should, in fact, be considered a cause and effect relationship.

The analytical elements that are shared by Sir Austin Bradford Hill's and the Surgeon General's methodologies include: the strength of the association; the consistency of the association among different studies; the specificity of the association; the temporal relationship of the onset of adverse health effect to the exposure(s) in question; and coherence, which involves the relationship of the agent to the natural history of the disease. In addition, Hill lists two other elements that have proven useful: whether a dose-response relationship exists, and the biological plausibility of the proposed cause and effect relationship. The most important of these generally accepted factors is consistency of statistically significant associations across well-conducted studies of different populations.

In order to employ this generally accepted approach correctly and test for consistency, one must adopt a "weight of the evidence" method, meaning that one must evaluate studies whether they are positive or negative. Thus, one must have reasonable confidence that he or she is obtaining, if not all relevant articles, at least enough to represent a fair cross-section, including both positive and negative findings. One cannot fairly assess the relevant literature on a causation issue by looking exclusively at positive findings, which does not allow one to address the issue of consistency among studies. Such a method constitutes "cherry picking" and is neither scientific nor a generally accepted methodology.

Before performing an analysis such as one described above, one must start with studies that show statistically significant findings. The importance of examining the "statistical significance" of associations cannot be overstated. As noted by the EPA (U.S. EPA, 2005), "The analysis should apply appropriate statistical methods to ascertain whether the observed associations between exposure and effects would be expected by chance." One method of describing statistical significance is the use of the confidence interval (CI), which identifies the range of results that would not occur due to chance. Almost all of the studies analyzed below include a 95% CI on the reported comparisons. Assessing whether a calculated rate is statistically significant (within the 95% confidence interval) is the generally accepted method used to reasonably rule out chance as a reason for the association.

A standardized mortality ratio (SMR) is a measurement used in cohort studies that divides the number of chemically-exposed persons who have died of a disease by the number expected to die from that disease in the general population.[3] Similarly, a standardized incidence ratio (SIR) involves the finding of a disease in a cohort. A SMR of greater than 1.0 is not statistically significant unless the lower number of the confidence interval is above 1.0. If the lower number of the confidence interval is 1.0, it is not considered to be statistically significant. For example, if the SMR is 1.5 and the 95% confidence interval is 0.8-2.0, the finding is not a statistically significant increase because the increase could be reasonably attributed to chance. If, however, the 95% confidence interval is 1.1-2.0, that is a statistically significant increase because it is unlikely that the true result is less than 1.0.

Even a statistically significant association will sometimes be found that is not valid. Spurious statistical associations between the disease being studied and the chemical in question can arise for many reasons, the most common being concurrent exposures, confounding effects due to improperly matched cases and controls, recall bias during interviews, and random associations

3 If a defined comparison group is used instead of the general population, the term relative risk (RR) is used instead of SMR. A similar statistical analysis called the odds ratio (OR) is used for case-control studies with similar considerations on statistical significance.

occurring by chance. Given that the usual CI is 95%, and even when the finding appears to be valid, it may be wrong 5% of the time through chance alone.

I also reviewed studies of workers exposed to benzene and the timeframe of their exposures compared to the onset of AML. Finally, I reviewed studies of workers exposed to refined benzene, including a group of rubber workers exposed to refined benzene, to examine the occurrence of AML at different exposure levels.

Studies of Occupational Exposures in Petroleum Refineries

The question of whether workers in the petroleum industry are at increased risk of AML has been studied in dozens of epidemiology studies. In some cases, new studies have updated previous studies on the same group of workers who have been followed for an additional time period. In many of the refineries studied, gasoline was the major petroleum product produced along with aviation fuel.

Studies of AML in 208,000 petroleum workers in the United States and United Kingdom were reviewed and included in a meta-analysis (Wong and Raabe, 1995; Raabe and Wong, 1996). Nineteen cohorts of workers were studied and statistically combined into this meta-analysis. Among the nineteen individual cohorts, the SMRs for AML ranged from 1.69 to 0. No studies reviewed had either a statistically significant deficit or increase in AML. When all nineteen cohorts were combined, the SMR was found to be 0.96 with 95% confidence limits (CI) of 0.81-1.13.

More recent mortality studies have confirmed the lack of association between AML and petroleum refinery workers. Some of these studies are updates of the studies included in the meta-analysis described above. Collingwood et al. (1996) in an update found an SMR of 0.95 (CI: 0.26-2.44) among workers at the Paulsboro, New Jersey Mobil refinery, which produces gasoline, aviation fuel, diesel fuel and lubrication oils. At the Beaumont, Texas Mobile refinery, a follow-up study reported an SMR of 1.36 (CI: 0.59-2.68) (Raabe et al., 1998) and another follow-up of this group found SMR of 1.47 (CI: 0.76-2.57) (Wong et al., 2001). The updated study of 28,840 Texaco workers (Divine et al., 1999) found an SMR for

AML of 1.29 (CI: 0.78-1.99). Updates of two Exxon refinery/petrochemical cohorts found AML rates of 1.52 (CI: 0.76-2.72) at Baton Rouge and at Baytown 2.13 (CI: 1.10-3.73) (Huebner et al., 2004). No increase in AML (SMR = 1.00; CI: 0.56-1.65) was found in U.S. Exxon employees surveyed between 1979 and 1992 (Huebner et al. 1997). The Chevron facilities in El Segundo and Richmond were updated (Satin et al., 2002) and the combined SMR=1.14 (CI: 0.66-1.82). A Gulf Oil Company Port Arthur facility study was updated (Satin et al., 1996) and found an SMR of 0.63 (CI: 0.30-1.15). Tsai et al. (2007) updated the study of the Shell Deer Park Manufacturing Complex finding SMRs of 0.95 and 1.10 (CIs: 0.60-1.42 and 0.65-1.73 among refinery and chemical workers, respectively).

In addition to the updates in the U.S., a study of 16,623 workers in the Australian petroleum industry (Health Watch, 2007) found no increase in cancer incidence for AML among male petroleum workers (SIR = 0.78; CI: 0.34-1.53) and a lower than expected AML mortality rate (SMR = 0.54; CI: 0.20-1.17). The update of the Canadian employees at Imperial Oil Limited found an SMR of 1.38 (CI: 0.83-2.16) (Lewis et al., 2000).

A pooled case-control study of petroleum workers from Canada, the United Kingdom and Australia was reported by Schnatter et al. (2012). This study combined three of the case-control studies described previously (Schnatter et al., 1996; Rushton and Romaniuk, 1997; Glass et al., 2003). This study reported no statistically significant increase for AML but did find an increased risk of MDS. Glass et al. (2014) later reported on the cases of myeloproliferative disease (MPD) in the pooled analysis. For MPD cases other than CML, none of the benzene exposure metrics showed a statistically significant association.

In summary, the overall petroleum refinery worker study data, which is extensive, show no increased risk of AML. One study found an increase in MDS, but not MPD. Although benzene is present in the various intermediate and final products that include gasoline and aviation fuels at these facilities, the levels of benzene are apparently not sufficient to produce an increased cancer risk that can be detected with reliable and generally accepted scientific methods. Only one of

these many studies found a statically significant increase, which is about the same as would be expected by chance from the 95% confidence level of the data.

Studies of Petroleum Products Including Exposures to Gasoline

A mortality cohort study of over 18,000 petroleum distribution workers who worked at land-based terminals or marine vessels between 1946 and 1985 did not find any increase in leukemia mortality (Wong et al., 1993). There was a decreased association with leukemia for both land-based terminal workers (SMR = 0.89; CI: 0.59-1.29) and for marine workers (SMR = 0.70; CI: 0.42-1.09). Duration of employment or exposure, age at first exposure or year of first exposure, cumulative exposure, peak intensity of exposure, and job category were not associated with increased risk of leukemia.

Rushton (1993) reported the results of an extension of follow up (1976 to 1989) of a cohort of workers employed for at least one year between 1 January 1950 and 31 December 1975 at oil distribution centers in Britain. Increases in AML were reported that were not statistically significant (SMR = 155; CI: 82-265). In a follow up case control study investigating the risk of leukemia associated with exposure to benzene in petroleum marketing and distribution workers in the U.K. was conducted by Rushton and Romaniuk (1997). There was some increase in ORs for all exposure groups compared to the lowest group, but none of these achieved statistical significance.

An extensive study was conducted of 19,000 service station workers in the Nordic countries exposed before 1970 and followed for 20 years (Lynge et al., 1997). No increase in overall leukemia was found (SMR = 0.9; CI: 0.6-1.3) and no statistically significant increase in AML was found (SMR = 1.4; CI: 0.8-2.4). This study included the previously studied Swedish service station workers in fuel retailing who were then followed for an additional 6 years (Jakobsson et al., 1993) in which an increase in AML was reported. Another study of over 27,000 filling station attendants in one region of Italy found 2 cases of leukemia instead of one expected and no cases in another region instead of 2 expected. Consequently, these studies of

gasoline workers (daily gasoline exposure) do not show increased risk of leukemia or AML.

An analysis of 14 cases of leukemia in Canadian petroleum distribution workers analyzed according to their benzene exposures, and no relationship was found between benzene exposures and leukemia (Schnatter et al., 1993; 1996). Another study of Canadian marketing and distribution workers reported an SMR of 1.32 for ANLL (CI: 0.49-2.88) and 1.20 for AML (CI: 0.39-2.80). An increased risk of leukemia was reported for Swedish men exposed to petroleum products in a case control study reported in a letter to the editor (Brandt et al., 1978).

Studies of Benzene Exposures and Acute Myelogenous Leukemia

Some cohort studies have found a statistically significant association between exposures to benzene and AML or acute non-lymphocytic leukemia (ANLL), which includes AML, acute monocytic leukemia and acute leukemias of "other and unspecified cell types" (Infante et al. 1977; Rinsky et al. 1981; 1987; 2002; Aksoy, 1985; Yin et al., 1989; Wong, 1995; Hayes et al., 1997; Costantini et al., 2003).

Rinsky et al. (1981; 1987; 2002) studied leukemia cases in the Pliofilm cohort, which is a process that used refined benzene to dissolve natural rubber, and this process exposed workers to high levels of benzene. This is the study of benzene exposures used by the U.S. EPA for risk assessment purposes (IRIS). This well documented study found an increased risk for leukemia (SMR = 2.47; CI: 1.38-4.07). An analysis of AML found a statistically significantly increased mortality from AML at higher but not lower exposure levels (Wong, 1995). A study of the incidence of hematologic neoplasms in China reported a statistically significant increase in leukemia (SMR = 2.5; CI: 1.2-5.1), in ANLL (SMR = 3.0; CI: 1.0-8.9) and ANLL/MDS (SMR = 4.1; CI: 1.4-11.6). This study was also reported by Yin et al. (1996), who reported that there were 7 cases of MDS in the benzene exposed workers compared to zero in the comparison group. Linet et al. (2015) provided further follow up through 1999 in this retrospective cohort. Significantly elevated incidence of all myeloid disorders included excesses of myelodysplastic syndrome/acute myeloid

leukemia (RR= 2.7; CI: 1.2-6.6), which included an additional case of MDS. This study by Hayes et al. (1997) will be discussed in more detail below.

Aksoy (1985) also reported a statistically significant increase in the mortality due to leukemia among Turkish shoe workers, but an SMR was not reported. A study of Italian shoe factory workers reported an increased mortality from leukemia in the higher but not lower exposure categories (Costantini et al., 2003). In these studies, no analysis of AML specifically was performed.

Other studies have not found benzene statistically significantly associated with leukemia, AML or ANLL. Some studies found less than the expected number of cases and others found more than expected. Wong (1987) published an industry wide mortality study of chemical workers occupationally exposed to benzene and found a less than expected rate of leukemia (SMR = 0.75; CI: 0.30-1.54). Fu et al. (1996) found fewer than expected leukemias (SMR = 0.89; CI: 0.51-1.45) in an English cohort and more than expected (SMR = 2.14; CI: 0.92-4.21) in an Italian cohort of shoe manufacturing worker exposed to benzene.

Bloemen et al. (2004) examined lymphohaematopoietic cancer risk among chemical workers exposed to benzene and found no increase for leukemia (SMR = 1.11; CI: 0.30-2.83). Another study found some non-statistically significant increases, but the numbers were small and the exposures were low (Collins et al., 2003). A study of benzene workers in England and Wales was completed by Sir Richard Doll (Sorahan et al., 2005). In their study, various industries including rubber, boots, shoes and leather, chemicals, petroleum and paints were investigated, and no statistically significant association between leukemia (SMR = 1.37; CI: 0.86-2.07) and benzene was found. A borderline statistically significant finding was found for ANLL (SMR = 1.83; CI: 1.00-3.07) and benzene exposure was found.

Wong et al. (2010) performed a hospital-based case-control study of acute myeloid leukemia in Shanghai. They analyzed environmental and occupational risk factors by subtypes of AML using the WHO (2001) classification. For all AML there was a statistically significant increase for exposures to benzene (OR = 1.43; 1.05-1.93). There were increased ORs for toluene and xylene that were

not statistically significant. The ORs for petroleum fuels, kerosene and gasoline were about as expected. For the classification of AML-MD defined as AML with multilineage dysplasia with or without preceding MDS, benzene exposures were not statistically significantly elevated (OR = 1.16; CI: 0.60-2.24). Petroleum fuels and gasoline (OR=1.07; CI: 0.72–1.61) were about as expected; kerosene was less than expected; and toluene and xylene were more than expected; but none of these finding were statistically significant.

Strom et al. (2012) reported a case-control study conducted at The University of Texas M. D. Anderson Cancer Center to investigate associations between lifestyle characteristics and the risk of AML in Texas. This study included 638 adult patients with de novo AML (cases) and a group of 636 matched controls. Questionnaires were used to collect demographic and occupational data. Among men, smoking 30 pack-years increased the risk of AML (OR = 1.86; CI: 1.15-3.02). Because the prevalence of exposure was low for individual chemicals, the investigators combined the exposures of benzene, gasoline, and other organic solvents as a measure of organic solvent exposure. They found an increased risk of AML for these solvent exposures, but there was no quantitation of the amount of benzene or other solvents.

In summary, one cohort and one case-control studies of workers exposed to high levels of benzene have shown increased mortality from AML. Another case-control study with unquantified benzene exposure also reported and increased risk of AML. Two other studies found a risk of leukemia and another where the risk was uncertain. Lower levels of exposure in these same studies found no increased risk. Other studies of workers exposed to benzene where dose was not specified found no increased risk. Therefore, as is the case with all drugs and chemicals, the dose of benzene is important in making a determination of causation.

Studies of Petroleum Products and Myelodysplastic Syndromes

In a study of Turkish shoe workers exposed to benzene, 51 cases of leukemia were found with 20 of these being described as AML (Askoy, 1985) but the majority would qualify as AML by today's definition. Of the 51 leukemia or preleukemia cases

identified by Askoy, 13 were preceded by pancytopenia, which would probably qualify as MDS. However, of 44 patients with pancytopenia, only 6 developed any form of leukemia, and we do not know what the expected rate of AML was in this cohort, i.e., the number of AML cases not caused by benzene.

Occupational and environmental risk factors for 400 cases of MDS in the U.K. were reported by West et al. (1995). High exposures for diesel fuel and gasoline or petroleum products in general did not significantly increase the risk of MDS (OR = 1.09; 1.25). A study of occupational and environmental risk factors of MDS in France reported increased odds ratios for solvents, oil, ammonia, pesticides, fertilizers, cereal dust, livestock, infective risk, cotton and flax dusts with the largest being for exposures to poultry (Nisse et al., 2001). However, none of these associations in the univariate analysis except for a risk of 1.1 (CI: 1.0-1.2) for exposures to oils remained when a multivariate analysis was performed. Risk factors for MDS including benzene exposure studied by Strom et al. (2005). There was a statistically significant increase for association of benzene/solvent/gasoline with MDS (OR=2.06; CI:1.14–3.71). It is not clear what "solvent" meant in this study.

Irons et al. (2005) have studied the dysplasia in the bone marrow associated with benzene exposure. They found that benzene exposure produced a unique type dysplasia that differs from commonly defined subtypes of MDS such as Mr. Webb's type. In a subsequent publication (Irons et al., 2010), Dr. Irons has analyzed 611 cases of MDS in China of which 80 had some exposures to benzene. Cases with definitive and high exposures to benzene were termed benzene signal cases defined as having exposures above 67 mg/m^3 (about 20 ppm) for an average of 12 years or 240 ppm-years. He reported that eosinophilic dysplasia was the most prevalent finding in benzene signal cases (MDS-Eo). I have seen no description of eosinophilic dysplasia in the reports of bone marrow biopsies for Mr. Webb. According to these authors the following:

> "The predominant pattern of BM pathology in BZ-exposed MDS cases, namely multilineage dysplasia, abnormal eosinophils, hematophagocytosis, together with stromal degeneration, is consistent with an ongoing inflammatory response persisting for many years after cessation of BZ exposure."

Among the cases diagnosed with RAEB, which is the type that was diagnosed for Mr. Webb by Dr. Harrison, there was no statistically significant association (OR = 1.4; CI: 0.19–11.1) for benzene exposure. For the unclassifiable form of MDS, which Mr. Webb did not have, there was an increased association in benzene signal cases (OR = 11.12; CI: 1.34-92.4).

Ruiz et al. (1994) reported the histological and cytological features of bone marrow in 152 employees from a steel plant in Brazil, who had been removed from areas of benzene exposure due to hematological abnormality. A similar type of dysplasia involving eosinophils was found in this earlier study. Repeated biological findings from persons exposed to high doses of benzene is significant. The fact that Mr. Webb did not have this feature is significant.

The Lv et al. (2010) and the Copley et al. (2017) publications reported case-control analysis of overlapping MDS cases from Shanghai hospitals. In the Lv et al. report, the highest risk of MDS was found for a lack of education (OR = 20.67; CI: 2.74-156.1). For all MDS, the risk of MDS was increased for benzene exposure (OR = 4.33; CI: 1.88-10.00). Statistically significant associations between benzene exposure and RCMD but not RAEB or other types of MDS were reported. There was no association between gasoline exposure and RAEB.

In the Copley et al. publication there were similar findings in that there were associations between benzene exposures for all MDS and RCMD. In addition, there was a dose response increase between benzene exposure and all MDS and RCMD but not for the RAEB type. However, the highest odds ratio was reported for the RCUD type in the highest exposure category (OR = 15.7; CI: 1.40-178). There was a statistically significant association for the higher categories of 0.3 to 3, 3 to 12 and above 12 ppm exposures over a several year period for these two types (ORs = 3.19, 5.38, 3.75) but not for less than 0.3 ppm. It is not clear how these levels compare to the usual metric of ppm-years used in other studies.

In another study, Poynter et al. (2017) reported the occupational and residential risk factors for cases of AML and MDS identified by the Minnesota Cancer Surveillance System. Exposure data were ascertained by a self-

administered questionnaire with a list of chemicals. There was an increase in greater than 5 years of benzene use for MDS cases (OR = 2.10; CI: 1.35-3.28). There was a statistically significant increase for MDS-U cases (OR = 4.52; CI: 1.20-17.0) but not for RAEB, RCMD, RARS or other types. For gasoline occupational and recreational exposure of greater than 5 years, there was no appreciable risk (OR=1.11; CI:0.71-1.74) compared to no exposure.

In summary, although benzene as a sufficient dose has been found to be associated with MDS, all studies except one show that this increase does not involved the RAEB type. Almost all studies implicate RCMD, RCUD, MDS-U but not RAEB, which was the type of MDS suggested in the medical record for identified by Dr. Harrison, being associated with benzene exposures. As will be discussed below, it is clear that sufficient doses of benzene can cause AML, and in some cases the AML has been preceded by MDS. The most prominent finding is the association of benzene exposure with MDS that involves eosinophilic dysplasia (Irons et al. 2010), which was not found in Mr. Webb.

Toxicological Effects of Concern Require a Significant Dose

The study of toxicology identifies potential health effects of chemicals and the dosage required to produce those effects. Without a sufficient dosage over a sufficient period of time, a chemical will not produce a toxic effect. As the first physician Paracelsus (1492-1541) who systematically studied occupational health and published his famous major work on the subject in 1567 stated, "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy." This statement is obvious for the description of medicines, where we know that a low dose can be therapeutic, but a higher dose can be toxic. However, the principle applies to other chemicals, and even though they do not have a beneficial effect at low doses, they will only have a toxic effect at high doses.

It should be noted that exposure only implies the opportunity for a chemical to be absorbed into the body. Sometimes the term "internal exposure" as opposed to "external exposure" is used. In any event, the presence of chemicals in the

workplace does not necessarily lead to internal exposure and, if it does, the amount may be very small.

Benzene exposure at sufficient dose can cause AML, and some of these cases have been reported to be preceded by MDS. However, as was discussed earlier in this report, the type of MDS that Dr. Harrison diagnosed for Mr. Webb with, i.e. RAEB, has not been described in the literature as occurring from benzene exposure.

Low Doses of Benzene Have Not Been Found to Cause AML

The study that has been cited most widely and is used by the U.S. EPA for risk assessment purposes is that of Rinsky et al., (1987). This is a study of rubber workers involved in the "Pliofilm" process, whereby natural rubber was dissolved in refined benzene and processed by being spread out on a conveyor belt system. Substantial exposures to refined benzene of over 400 ppm-years[4] resulted in this work environment with the occurrence of AML in workers. In this study (Rinsky et al., (1987), workers with exposures of less than 40 ppm-years showed no excess of total leukemia compared to the general population. Although Rinsky reported that there was an increase in leukemia above this exposure level, a statistically significant increase was only found above 200 ppm-years. Also, when the data from this study was analyzed for only cases of AML, there was no increased risk until exposures exceeded 200 ppm-years (Wong, 1995).

In a study of Chinese workers in various industries exposed to benzene, no statistically significant increase in MDS and ANLL cases was reported below 40 ppm-years (Hayes et al., 1997). Another study of the dose-response of benzene exposure in Italian shoe workers found results similar to these studies (Costantini et al., 2003). No appreciable increase was found at less than 40 ppm-years, and no statistically significant increase was found below 200 ppm-years.

4. A ppm-year is the air concentration in parts per million averaged over an 8 hour work day for a working year multiplied by the number of years of exposure.

The group of workers where no increase in ANLL was reported (Health Watch, 2005; 2007) also reported finding a statistically significant increase in leukemia above 8 ppm-years benzene exposure (Glass et al., 2003; 2005). Terminal workers who would have distributed gasoline showed about the number of AML cases (SIR=1.11; CI: 0.36 - 2.60). This the same study based on information in the report by Gray et al. (2001). However, the reason for the reported effect above 8 ppm-years is that the analysis by Glass et al. (2003; 2005) and by Gray et al. (2001) involves comparison with a lowest exposure group in which the ANLL incidence was unexpectedly low. On page 69 of Gray et al. report, one only needs to look at the difference between Tables 35 and 36 to understand why there were erroneously increases for leukemia but not lymphatic cancer in all except in the highest dose group. Table 36 shows that in the lowest exposure group, there were 0.05 cases of leukemia for each of the controls. However, since there were 4 controls selected for each case, there should be 0.25 cases of leukemia for each of the controls where benzene had no effect. Therefore, this group for whatever reason has 20% of the cases than expected. This unexpectedly low incidence, which is used for comparison to all the other groups, creates relative risks about 5 times higher than should be the case, and this partly explains the dose-response information that is different from that in the Rinsky et al. (1987), Hayes et al. (1997) and Costantini et al. (2003) publications. This highest dose group contained workers exposed from 16 to 57 ppm years. Table 43 also shows that the increase for AML and ANLL is only in the highest dosage group; however, given the problems with the low rate in the 1-3 quintiles, it is not clear that the highest quintile would be statistically significant. In contrast for lymphatic cancers in Table 35, there were 0.19 cases in the lowest exposure group, close to the expected 0.25 cases.

A case-control study investigating the risk of leukemia associated with exposure to benzene in petroleum workers in the U.K. was conducted by Rushton and Romaniuk (1997). There was some increase in ORs for all exposure groups compared to the lowest group, but none of these achieved statistical significance. Also, as was seen in the Australian study cited above, the number of cases compared to controls was low when corrected for study design (24 cases versus 35

controls), which accounted for some of the increase in the other groups.

Low Doses of Benzene Have Not Been Found to Cause MDS

When benzene exposure levels were historically much higher than at present, there were many reports of aplastic anemia, preleukemia, and other hematologic conditions that resemble MDS. However, the occurrence of these conditions has been described much less frequently in the later 1900s as the levels of benzene in the workplace have decreased. More recently as better diagnostic criteria have been developed, more cases of MDS have been reported and differentiated from AML.

The studies of the dose-response of benzene with the development of AML described previously also provide information regarding the dose response of MDS since many of these AML cases would have been preceded by MDS. Therefore, the studies by Rinsky et al. (1987) and Wong (1995) previously described for leukemia also has bearing on the understanding of the dose-response for benzene and the development of MDS. In these studies, there was no statistically significant increase in total leukemia below 40 ppm-years or in AML below 200 ppm-years.

The study of Chinese workers exposed to benzene, and MDS cases were included in one of the dose-response analyses (Hayes et al., 1997). Risk for ANLL/MDS increased with increasing cumulative exposure to benzene: cumulative ppm-years: 40-99, (RR= 6.0; CI:1.8-20.6) and cumulative ppm-years: >100 (RR=4.4; CI:1.4-13.5). For less than 40 ppm-years the risk was not statistically significantly elevated.

A pooled case-control study of petroleum workers from Canada, the United Kingdom and Australia was reported by Schnatter et al. (2012).[5] This study combined three of the case-control studies described previously (Schnatter et al., 1996; Rushton and Romaniuk, 1997; Glass et al., 2003). Different types of MDS were not analyzed in this study. This study reported that exposures of greater than

5. Also included is the report dated February 15, 2012, which is included in the Reference List.

2.93 ppm-years caused a statistically significant increase in MDS but not of AML. In fact, there was no statistically significant increase at all for AML reported in this study indicating that the study does not show what other studies have reported, i.e., that benzene exposures increases the risk of AML. Also, this study did not differentiate between the various type of MDS. Remarkably, in this study the highest risk is shown for CML (which is not caused by benzene or gasoline) in the mid-exposure group, but not in the high exposure group. This shows the instability of that statistics reported in this study. The results of this study suffer from the same difficulty as in the case of the previous Australian study. There were approximately 4 controls chosen for each case. However, for MDS, the referent group to which the low and high exposures were compared contained about 8 controls. In this study, smoking was associated with MDS; however, there was incomplete smoking data.

Because of the lack of reliable information regarding dose-response for MDS, the dose-response information for AML from the Rinsky et al. (1987) study and for ANLL/MDS from the Hayes et al. (1997) study become the most important dose-response data to compare with Mr. Webb 's exposures. These studies show no statistically significant increase in risk below 40 ppm-years, and in the Rinsky et al. study, no statistically significant increase below 200 ppm-years.

Further confirmation that low levels of exposure to benzene do not cause MDS come from a study by Sorahan and Mohammed (2016), who reported the incidence of myelodysplastic syndrome in UK petroleum distribution and oil refinery workers from 1995-2011. The incidence of myelodysplastic syndrome (MDS) experienced by cohorts of 16,467 petroleum distribution workers and 28,554 oil refinery workers was investigated. Study subjects were all those male employees first employed at one of 476 UK petroleum distribution centers or eight UK oil refineries in the period 1946–1974; all subjects had a minimum of twelve months employment with some employment after 1st January, 1951. Observed numbers of MDS cases were compared with expectations based on national incidence rates for 1995–2011. The overall SRR findings did not provide clear evidence f of an occupational cancer hazard, and provide no support for the hypothesis that low-level benzene exposure has an important effect on the risks of MDS.

Biological Plausibility of a Threshold for Gasoline

The explanation for why benzene at low doses does not cause AML or MDS involves the mechanism by which benzene has its genotoxic effects and an understanding of how other chemicals could modulate these effects. The mechanism of genotoxicity caused by benzene can be causally linked to inhibition of topoisomerase II, an enzyme that is necessary for the proper segregation of chromosomes during cell replication. Inhibitors of this enzyme produce translocations and deletions of chromosomes. I have reviewed all available studies of benzene genotoxicity (Whysner, 2000, Whysner et al., 2004), and benzene was found to produce evidence of clastogenicity (translocations and deletions) but not mutagenicity. The clastogenicity was found to be best explained by inhibition of topoisomerase II as a mechanism, which has been found to have a threshold dose (Lynch et al., 2003; FDA, 2006).

Thresholds have also been proposed for other types of genotoxic carcinogens. Studies of formaldehyde have shown that carcinogenicity requires doses in excess of thresholds for several toxic effects in addition to genotoxicity (Conaway et al., 1996). Studies of mutagenicity for carcinogens have been found to have thresholds due to DNA repair enzymes (Sofuni et al., 2000). Thresholds have been found for the experimental liver carcinogen a-acetylaminofluorene (Williams et al., 2004). A recent ECETOC workshop reported the results of an expert panel on the biological significance of threshold for effects produced by genotoxic carcinogens (Pottenger et al., 2009).

Another explanation for the lack of carcinogenicity of low doses of benzene in gasoline involves the interactions of chemical mixtures. Chemicals such as gasoline cannot be evaluated scientifically in the absence of understanding the potential competing effect of components in the mixture. This is especially true for mixtures containing smaller amounts of benzene than other components. The inhibition of the metabolism of benzene to its toxic metabolites is caused by toluene, which is a common constituent of chemical mixtures containing small amounts of benzene

(Medinsky et al., 1994). Inoue et al. (1988) found that the toxic metabolites of benzene, phenol, and hydroquinone were much lower in workers also exposed to toluene. Other evidence of suppression of benzene toxicity in mixed exposures with toluene is found in genotoxicity tests. Toluene reduced the number of sister chromatid exchanges induced by benzene when both compounds were administered intraperitoneally to DBA/2 mice (Tice *et al.*, 1982), and it greatly reduced the frequency of micronuclei induced by benzene when the two compounds were simultaneously administered orally to CD-1 mice (Gad-El-Karim *et al.*, 1984), intraperitoneally to Sprague-Dawley rats (Roh *et al.*, 1987) or subcutaneously to NMRI mice (Tunek *et al.*, 1982), but not when intermittent exposures were given to CD-1 mice (Wetmore et al., 2008). Therefore, the available scientific evidence suggests that other constituents of petroleum products may reduce the toxicity including genotoxicity and carcinogenicity of benzene (as found in a mixture).

Review of Mr. Webb's Medical and Employment History

Mr. Webb was 65 years old when on 1/12/2015 when a bone marrow biopsy taken due Mr. Webb's chronic leukocytosis, and anemia. The findings in the bone marrow were not found in the medical record. On 8/24/2015 another bone marrow biopsy was performed and reported the next day. The bone marrow was found to be hypercellular (too many cells) with evidence of a myeloproliferative disorder. The differential count revealed no increase in eosinophils at 0.6%. No BCR-ABL1 transcripts were detected. The peripheral blood revealed a white count of 24,600 per cubic mm and the platelet count was normal. A progress note on 11/11/2015 reported that oncology was consulted and Mr. Webb was considered to have a myeloproliferative disorder. A blood test on 11/20/2015 showed thrombocytopenia along with anemia and a normal total WBC. Monocytes were low and myelocytes were high.

A third bone marrow on 1/12/2016 reported marked hypercellularity with trilineage dyspoiesis and noted that, "The overall findings are most consistent [with] a myelodysplastic/myeloproliferative neoplasm." A cytogenetic analysis

showed a normal karyotype. Subsequently, the oncologist Dr. Chandra considered the clinical picture as suspicious of MDS, but did not specify a type.

Mr. Webb was born on December 23, 1950. Deposition testimony of Mary Major reports that Mr. Webb smoked from before 1982, which was when she first knew him, until 2008. The medical records in 2006 and 2009 noted that Mr. Webb smoked at least a pack of cigarettes per day. There were several notes in the chart where he was urged to quite because of bronchitis or wheezing. Therefore, his smoking history is somewhere between 25 and 30 pack-years. In 2013, the record indicates that he had quit smoking in 2009 after 30+ years of tobacco use.

Mr. Webb's height was reported in the medical chart to be 5 feet 7 inched. According to Mary Major, on page 37 of her deposition, his weight was 220 lbs. This would give him a BMI of 34.5, which would be classified as obese. On 7/5/2015, the medical record reports that his weight was 99.5 kg, which would be 219 lbs.

Other problems noted in the medical record were abdominal aortic aneurysm, kidney stones, gout and diverticulosis. Family history reported various cardiovascular problems, hypertension and diabetes.

From approximately 1985 to 2016, Mr. Webb worked as truck driver for Calzona Tankways and later Coastal Transport Company, Inc. transporting gasoline, avgas, and diesel fuel.

Cigarette Smoking and AML

There is strong evidence that cigarette smoking is associated with AML. The most recent report by the Surgeon General of the U.S. Public Health Service (2004) entitled "The Health Consequences of Smoking" noted the following in its "Conclusions" on p. 254:

> "1. The evidence is sufficient to infer a causal relationship between smoking and acute myeloid leukemia.
> 2. The risk for acute myeloid leukemia increases with the number of cigarettes smoked and with duration of smoking."

Additionally, this document reports the following:

> "A relationship between former or current smoking and the risk of acute myeloid leukemia is supported by evidence of a consistent dose-response relationship with the number of cigarettes smoked per day. The association of the duration of smoking with the degree of risk and an increase in risk among former smokers suggests that the relationship is not dependent on current smoking, but perhaps on the cumulative effects of cigarette smoking."

Colamesta et al. (2016) performed the most recent meta-analysis on the relationship between tobacco smoking and the onset of acute myeloid leukemia (AML) in adults, which included twenty-seven articles. Case-control and cohort meta-analyses showed that current, ever and former smokers have a significant increased risk to develop AML compared to never smokers. Moreover, increasing smoking intensity and duration is associated with an increase of the risk of AML. For a smoking history of 30 pack-years, there was a doubling of the AML risk.

Cigarette Smoking and MDS

Ma et al. (2009) examined the relation of smoking to MDS in a cohort of 471,799 persons aged 50–71 years who were recruited into the National Institutes of Health-AARP Diet and Health Study, a large US prospective study, in 1995–1996. The risk of MDS was elevated among former smokers (hazard ratio = 1.68; CI: 1.17-2.41) and current smokers (hazard ratio = 3.17; CI: 2.02-4.98) as compared with never smokers. For former smokers of greater than one pack-per-day the hazard ratio was 1.87 (CI: 1.23-2.83).

Obesity and AML or MDS

Obesity has also been associated with leukemia. Mr. Webb's body mass index (BMI) would have been 34.5 according to the testimony of Mary Major. The study by Calle et al. (2003) found that men with BMI's above 35 were at a statistically significantly increased risk of leukemia (RR= 1.37; CI:1.13–1.67). In another study by the National Cancer Institute of over 4 million veterans, obesity was associated with AML (Samanic et al., 2004).

Ma et al., 2009 examined the relations of obesity and lifestyle factors to MDS in a cohort of 471,799 persons aged 50–71 years who were recruited into the National Institutes of Health-AARP Diet and Health Study, a large US prospective study, in 1995–1996. A significant positive association was observed between BMI and MDS (P for trend < 0.001). The hazard ratios for persons with BMIs of > 30 was 2.18 (CI: 1.51-3.17).

Summary of Mr. Webb's Risk Factors for His Alleged MDS

Mr. combined risk factors of obesity and smoking result in an increased risk of MDS/AML of greater than two and perhaps three-fold.

Mr. Webb's Benzene Exposures and the Development of His Alleged MDS

The question of the importance of recent versus distant exposures has been studied with regard to benzene and the development of leukemia. Several studies have found that exposures less than 15 years before the development of AML or MDS are the only ones that present a risk for these conditions.

Several analyses of the Pliofilm cohort have also examined the issue of time of exposure. This group of workers was exposed to benzene until 1965 at one plant and until 1976 at another. No additional deaths from leukemia were reported 15 years after the closing of the plant in 1976 (Rinsky et al., 2002). Silver et al. (2002) analyzed "exposure windows" for benzene in the follow-up of the cohort through 1996 reported by Rinsky et al. (2002). They each worker's cumulative exposure that was split out into exposure windows of 0–4.9, 5–9.9, 10–14.9, and 15 years or more prior to the cutoff (age at death of the case). They found that exposures more than 15 years prior to death from leukemia did not contribute to the risk. It is clear that since leukemia could be diagnosed prior to death, this exposure interval would be even less than 15 years for the diagnosis of leukemia.

Finkelstein (2000) analyzed data for the earlier follow-up through 1987 on this cohort (Rinsky, 1987) including additional cases provided by NIOSH after this publication. He stated that, "The coefficients for those windows more than 14 years before the death of the case were not statistically significant; that is, there were no

significant differences between the exposures of the case subjects and their controls more than 14 years before the death of the case."

A study of Chinese workers by Hayes et al. (1997) compared death rates in factory workers exposed to benzene compared to unexposed workers. These authors have examined the issue of time of benzene exposure in relation to death from ANLL/MDS and stated the following:

> "In our study, ANLL/MDS was linked to recent benzene exposure, and additional distant exposure did not appear to further increase risk. This finding suggests that recent benzene exposure is predictive of subsequent risk for ANLL/MDS, analogous to the short latency and wavelike increase then decrease in risk seen for several forms of radiation-induced leukemia (19) and for chemotherapy-related AML (18)."

In this study the difference between recent and distant exposure was 10 years.

Also, in the evaluation of its cohort of Australian petroleum workers, Glass et al., (2004) reported similar findings although cases included newly diagnosed workers as well as deaths. These authors stated the following:

> "We found that leukemia was most strongly associated with benzene exposures in the period 15 years prior to diagnosis and exposures more than 15 years prior to diagnosis made little contribution to the risk."

Wong et al. (2010) also analyzed exposure time in their hospital-based case-control study of acute myeloid leukemia in Shanghai. These investigators also found that only exposures prior to the year 2000 were statistically significantly associated with AML (OR=4.18; CI:1.56–11.15). The cases in this study were hospital-based case-control study consisting of 722 newly diagnosed AML cases from August 2003 through June 2007. Therefore, the greatest risk was 7 years prior to diagnosis.

Triebig (2010a) presented the data of 537 confirmed cases of leukemia as an occupational disease in Germany during the time period 1978–2007. He concluded that the epidemiologic findings demonstrated a smaller or even absent risk of leukemia 10–15 years after exposure to benzene has been stopped. Triebig also

reviewed the results of several independent epidemiologic studies of occupational cohorts with benzene exposure described previously. He concluded that temporal changes in relative risk highlights the importance of examining the relationship between follow-up time and risk estimates as part of the risk assessment process.

In a letter to the editor by Straube of a "comment on" the study by Triebig (2010a) describes studies of rats given nitrosamines in the drinking water and their development of esophageal and liver to assert that DNA damage of carcinogens is irreversible. However, Straube acknowledges that DNA repair can take place. In Triebig's response to the Straube comment (Triebig, 2010b) he stated the following:

> "The fact that latency periods play an important role in carcinogenicity has been shown for a majority of various chemical and physical factors. For example, the high latency period in the case of smoking and the development of lung as well as bladder cancer or the effect of radiation and lung cancer are scientifically well established."

Furthermore, the review of information by Bhatia (2013) describes the effects of chemotherapeutic agents that work by damaging DNA cause secondary cancers such as leukemia within 7 years. Consequently, either the damage is repaired or the damaged cells die within this time period. The concept that cells could be damaged, become quiescent, and then later cause cancer is contrary to the medical literature about these DNA damaging agents. Chemotherapeutic agents are designed to kill rapidly dividing cancer cells, not to put them to sleep for some period.

Besides the studies described above for benzene, this window of exposure for the development of AML or MDS is supported by studies of benzene metabolism and the development of AML and MDS from chemotherapeutic agents. Exposures by to benzene can leave the body rapidly through inhalation and metabolism. Most of the metabolites of benzene leave the body in the urine within 48 hours after exposure (ATSDR, 2007). As reviewed by Bhatia (2013), the time of exposure and date of diagnosis for chemotherapeutic agent can be accurately studied since the date that the exposure is known, and patients are followed regularly to detect the onset of MDS or AML. Two types of MDS or AML occur depending on the causative

therapeutic exposure: an alkylating agent/radiation-related type and a topoisomerase II inhibitor-related type. Alkylating agent-related MDS or AML usually appears 4 to 7 years after exposure to the mutagenic agent. Approximately two thirds of patients present with MDS and the remainder with AML with myelodysplastic features. The latency period between the initiation of treatment with topoisomerase II inhibitors and the onset of leukemia is less, with a median of 2 to 3 years.

Therefore, for purposes of comparison to studies of benzene exposure and diagnosis or death from MDS, exposures 10 to 15 years prior to his diagnosis would be relevant, which would not include any of his exposures hauling gasoline or other petroleum products. The dose-response information in the major studies of benzene exposures and the development of AML/MDS show that greater than 40 ppm-years is required to increase the risk of AML/MDS. The calculated values by Mr. Spencer of Mr. Webb's reasonable worst-case cumulative dose from exposure to benzene from his use of all products 15 years prior to his diagnosis ranged from 0.26 ppm-years to 0.79 ppm-years, and for Mr. Webb's entire career 0.54 ppm-years to 1.6 ppm-years. This benzene exposure is not sufficient to conclude that Mr. Webb's alleged MDS was caused by benzene exposures, even using the dose-response information in the study by Schnatter et al. (2012), which I have previously found to be flawed. Additionally, MPN has not been causally associated with benzene exposures (Schnatter et al. 2012) and the one form of MDS/MPN studied was not found to be associated with benzene exposures (Gross et al., 2012).

The exposure assessment performed by Dr. Jones is inadequate for determining Mr. Webb's risk for his alleged MDS because she didn't take into account the relevant window of exposure. Even so, the ppm-years calculated by Dr. Jones do not prove that Mr. Webb's MDS was due to benzene exposures. Furthermore, gasoline and the other identified petroleum products have not been found to cause MDS, AML, MPN or MDS/MPN.

Evaluation of Dr. Harrison's Report Regarding Causation

Mr. Webb was diagnosed with "myelodysplastic/myeloproliferative neoplasm" based on the pathology report from a bone marrow examination collected in January 16, 2016: however, according to the complaint and Dr. Harrison's on page 7 of his report the alleged diagnosis was "Myelodyplastic syndrome MDS/RAEB-1." Harrison's general causation assessment does not account for the type of MDS identified by Dr. Harrison, i.e., RAEB, which has not been found to be caused by benzene exposure. He states the following:

> "The literature upon which I rely is appended to this report, including several recent papers regarding benzene and the increased risk of MDS (Schnatter 2012, Sorahan 2016, Copley 2017, Poynter 2017, Li 2018)."

As described previously in this report, the studies by Copley and Poynter found that benzene exposures were associated with some types of MDS, but not the RAEB type. The other studies by Schnatter and Sorahan relied upon by Harrison did not examine MDS types, and the study by Li was a review and did reach a conclusion regarding MDS types.

Harrison did not provide any studies for dose-response to compare with the exposure calculations given by Gilmore. Harrison opines the following:

> "In addition to my *qualitative* judgment that Mr. Webb was exposed to significant amounts of benzene that were sufficient to cause his MDS, Dr. Jones estimates that Mr. Webb's cumulative exposure to benzene through the inhalation and dermal route was as high as 13.1 ppm- years."

Also, he provides a "qualitative" assessment without any justification other than his statement in his summary as follows:

> "A qualitative analysis establishes that exposure to these products were substantial and were significant contributing factors in causing his MDS."

Instead Harrison cites the following from NIOSH:

> As stated by NIOSH: "Underlying this policy is the recognition that there is no known safe level of exposure to a carcinogen, and therefore that reduction of worker exposure to chemical carcinogens as much as possible through elimination or substitution and engineering controls is the primary way to prevent occupational cancer."

It should be noted that a regulatory agency such as NIOSH aims to be health protective rather than determining at what levels risks of disease actually occur. In fact, there are levels in studies that show no increase in AML or MDS and benzene exposures, and I have described these previously.

Dr. Harrison Does Not Provide an Adequate Analysis of Mr. Webb's Cigarette Smoking and His Risk of MDS from Smoking

Dr. Harrison cites only the deposition of Mary Major for Mr. Webb's smoking history in which Dr. Harrison reports states that he smoked from 1982 until 2008. However, Also, there is additional information in the medical record. For example, the record of an admission to Banner Estrella Medical Center in 2009 states that Mr. Webb smoked at least a pack of cigarettes per day. Also, there are numerous notes during this admission of admonitions for quitting smoking because of Mr. Webb's respiratory problems such as wheezing.

Dr. Harrison does not discuss Mr. Webb's smoking history in relation to the scientific and medical literature that he includes in his reference list. Therefore, he does not provide an adequate analysis of smoking as a risk factor for the development of Mr. Webb's alleged MDS.

Dr. Harrison Does Not Mention Mr. Webb's Obesity

Dr. Harrison inaccurately cites Mary Major's deposition testimony about Mr. Webb's smoking history. However, he does not report the testimony by Mary Major regarding Mr. Webb's weight that would classify him as being obese.

Summary

After reviewing the medical and scientific literature, I have found that it has not been established on the basis of relevant and reliable methodology that gasoline, avgas or other petroleum products of the type identified in this case are capable of causing MDS or AML. The type of MDS with which Dr. Harrison identified for Mr. Webb, i.e., RAEB, has not been found to be causally associated with benzene exposure. I have also found that it has not been established on the basis of relevant

and reliable methodology that there was sufficient benzene exposure to cause or contribute to Mr. Web's alleged MDS. Most of his exposure would have occurred prior to the relevant window of exposure for the development of MDS or AML, which would exclude some exposures as a gasoline and avgas truck driver. Mr. Webb was a cigarette smoker, which is causally associated with AML and MDS. His increased risk due to these factors was greater than two-fold. In addition, Mr. Webb was obese, which further increased his risk for the development of MDS. Plaintiff's expert Harrison did not provide an adequate basis for general causation for the products at issue to cause MDS, and in particular Mr. Webb's alleged type of MDS. He also did not establish any analysis of the cumulative exposure of benzene for any of Mr. Webb's benzene exposure, nor for the 15 years prior to his diagnosis, which is the relevant time period. Dr. Harrison's evaluation of the causal relationship between smoking with the alleged development of Mr. Webb's MDS was inadequate, and he did not mention Mr. Webb's obesity. All of my opinions in this case are within a reasonable degree of medical/scientific certainty.

July 22, 2019
Dated

John Whysner MD, PhD, DABT

References

ACGIH. 2001. Gasoline. Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists.

ACGIH. 2014. Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists.

Acquavella JF, Wiggs LD, and Wilkinson, GS. 1985. Mortality among workers at the Pantex Weapons Facility. Health Physics 48:735-746.

Adegoke OJ, Blair A, Shu XO, Sanderson M, Jin F, Dosemeci M.et al. 2003. Occupational history and exposure and the risk of adult leukemia in Shanghai. Ann Epidemiol 13:485-494.

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Gasoline. U.S. Dept. of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for JP-5 and JP-8. U.S. Dept. of Health and Human Services.

ATSDR (Agency for Toxic Substances and Disease Registry). 2007. Toxicological Profile for Benzene. U.S. Dept. of Health and Human Services.

Aksoy M. 1985. Malignancies due to occupational exposure to benzene. American Journal of Industrial Medicine 7:395-402.

Arp E, Wolf P. 1983. Lymphocytic Leukemia and Exposures to Benzene and Other Solvents in the Rubber Industry. JOM 25(8): 598-602.

Bhatia S. 2013. Therapy-related myelodysplasia and acute myeloid leukemia. Semin Oncol. 40:666-75.

Blair A, Hartge P, Stewart PA, McAdams M, Lubin, J. 1998. Mortality and cancer incidence of aircraft maintenance workers exposed to trichloroethylene and other organic solvents and chemicals: extended follow-up. Occup Environ Med 55:161-171.

Bloemen L, Youk A, Bradley T, et al. 2004. Lymphohaematopoietic Cancer Risk Among Chemical Workers Exposed to Benzene. Occup Environ Med. 61: 270-274.

Brandt L, Nilsson PG, Mitelman P. 1978. Occupational exposure to petroleum products in mem with acute non-lymphocytic leukaemia. British Med. J. 1:553.

Brunning RD, Matutes E, Harris NL, Flandrin G. et al. 2001. Acute myeloid leukemia: Introduction. Pathology and Genetics: Tumors of the Haematopoietic and Lymphoid Tissues. World Health Organization. Jaffe ES, et al., Editors. IARC Press. Pp. 77-87.

Brunning RD, Orazi A, Germing U. et al. 2008. Myelodysplastic syndromes/neoplasms, overview. WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues. World Health Organization. Swerdlow, et al., Editors. IARC. Pp. 88-93.

Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. 2003. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. New Engl J Med 348(17):1625-1638.

Carpenter AV, Flanders WD, Frome EL, Tankersley WG, Frey SA. 1988. Chemical exposures and central nervous system cancer: a case-control study among workers at two nuclear facilities. Am J Ind Med 13:351-362.

Colamesta V, D'Aguanno S, Breccia M, Bruffa S, Cartoni C, La Torre G. 2016. Do the smoking intensity and duration, the years since quitting, the methodological quality and the year of publication of the studies affect the results of the meta-analysis on cigarette smoking and Acute Myeloid Leukemia (AML) in adults? Crit Rev Oncol Hematol. 99:376-88.

Collingwood KW, Raabe GK, Wong O. 1996. An updated cohort mortality study of workers at a northeastern United States petroleum refinery. Int Arch Occup Environ Health 68(5):277-288.

Collins JJ, Ireland B, Buckley CF, Shepperly D. 2003. Lymphohaematopoeitic cancer mortality among workers with benzene exposure. Occup Environ Med 60:676-679.

Conaway CC, Whysner J, Verna LK, et al. 1996. Formaldehyde mechanistic data and risk assessment: Endogenous protection from DNA adduct formation. Pharmacol. Ther. 71:29-55.

Copley GB, Schnatter AR, Armstrong TW, Irons RD, Chen M, Wang XQ, Kerzic P. 2017. Hospital-Based Case-Control Study of MDS Subtypes and Benzene Exposure in Shanghai. J Occup Environ Med. 59:349-355.

Costantini AS, Quinn M, Consonni D, Zappa M. 2003. Exposure to benzene and risk of leukemia among shoe factory workers. Scand J Work Environ Health 29:51-59.

Costantini AS, Benvenuti A, Vineis P., et al. 2008. Risk of leukemia and multiple myeloma associated with exposure to benzene and other organic solvents: Evidence from the Italian multicenter case-control study. Am J Ind Med 51:803-811.

Delzell E, Monson, RR. 1981. Mortality among rubber workers III. Cause-specific mortality, 1940-1978. JOM 23:677-684.

Divine BJ, Hartman CM, Wendt JK. 1999. Update of the Texaco mortality study 1947-93: Part II. Analysis of specific causes of death for white men employed in refining, research, and petrochemical workers. Occup Environ Med 56(3):174-180.

FDA. 2006. Guidance for Industry and Review Staff. Recommended Approaches to Integration of Genetic Toxicology Study Results. U.S. Food and Drug Administration.

Finkelstein MM. 2000. Leukemia after exposure to benzene: temporal trends and implications for standards. Am J Ind Med. 38:1-7.

Fu H, Damers P, Costantini A, et al. 1996. Cancer mortality among shoe manufacturing workers: An analysis of two cohorts. Occup Environ Med. 53:394-398.

Gad-El-Karim MM, Harper BL, Legator MS. 1984. Modifications in the myeloclastogenic effect of benzene in mice with toluene, phenobarbital, 3-methylcholanthrene, Aroclor 1254 and SKF-525A. Mutat Res 135(3):225-243.

Gerin M, Siemiatycki J, Desy M, Krewski D. 1998. Associations between several sites of cancer and occupational exposure to benzene, toluene, xylene and styrene: results of a case-control study in Montreal. Am J Ind Med 34:144-156.

Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR, Fritschi L, et al. 2003. Leukemia risk associated with low-level benzene exposure. Epidemiology 14(5):569-577.

Glass DC, Sim MR, Fritschi L, Gray CN, Jolley DJ, Gibbons C. 2004. Leukemia risk and relevant benzene exposure period-Re: follow-up time on risk estimates, Am J Ind Med 42:481-489, 2002. Am J Ind Med. 2004 45:222-3

Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR. 2005. Health Watch exposure estimates: do they underestimate benzene exposure? Chem Biol Interact 153-154:23-32.

Glass DC, Schnatter AR, Tang G, Irons RD, Rushton L. 2014. Risk of myeloproliferative disease and chronic myeloid leukaemia following exposure to low-level benzene in a nested case-control study of petroleum workers. Occup Environ Med. 71:266-74.

Gray CN, et al. 2001. Lympho-haematopoietic Cancer and Exposure to Benzene

in the Australian Petroleum Industry. Monash University and Deakin University:221 p.

Gross SA, Irons RD, Scott PK, Galbraith D, Wang XQ, Chen Y, Paustenbach D. 20012. A case-control study of chronic myelomonocytic leukemia (CMML) in Shanghai, China: evaluation of risk factors for CMML, with special focus on benzene. Arch Environ Occup Health. 67:206-18.

Hayes RB, Yin SN, Dosemeci M, Li GL, Wacholder S, Travis LB, et al. 1997. Benzene and the dose-related incidence of hematologic neoplasms in China. Chinese Academy of Preventive Medicine—National Cancer Institute Benzene Study Group. J Natl Cancer Inst 89(14):1065-1071.

Health Watch. 2005. The Australian Institute of Petroleum Health Surveillance Program: Twelfth Report 2005. Department of Public Health, University of Adelaide, South Australia. pp:1-82.

Health Watch. 2007. The Australian Institute of Petroleum Health Surveillance Program: Thirteenth Report 2007. Department of Public Health, University of Adelaide, South Australia. pp:1-87.

Hill AB. 1965. The Environment and Disease: Association or Causation? Proceed Royal Soc Med 58:295-300.

Holmberg B, Westerhold, P, Maasing R, et al. 1983. Retrospective cohort study of two plants in the Swedish rubber industry. Scan J Work Environ Health 9(suppl 2):59-68.

Huebner WW, Schnatter R, Nicolich, MJ, Jorgensen G. 1997. Mortality experience of a young petrochemical industry cohort. J Occup Environ Med 39(10):970-982.

Huebner, WW, Wojcik NC, Rosamilia K, Jorgensen G, Milano CA. 2004. Mortality updates (1970-1997) of two refinery/petrochemical plant cohorts at Baton Rouge, Louisiana, and Baytown, Texas. J Occup Environ Med 46(12):1229-1245.

IARC. 1974. Some Anti-Thyroid and Related Substances, Nitrofurans and Industrial Chemicals. Benzene. Monographs on the Evaluation of Carcinogenic Risks to Humans. 7:203-221.

IARC. 1982. Some Industrial Chemicals and Dyestuffs: Benzene. Monographs on the Evaluation of Carcinogenic Risks to Humans 29:93-148.

IARC. 1989a. Gasoline. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. Monographs on the Evaluation of Carcinogenic Risks to Humans 45:159-201.

IARC. 1989c. Jet fuel. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels: Gasoline. Monographs on the Evaluation of Carcinogenic Risks to Humans 45: 203-218.

IARC. 1989d. Diesel Fuel. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels Monographs on the Evaluation of Carcinogenic Risks to Humans 45:159-201.

IARC. 1999a. Toluene. Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide. Monographs on the Evaluation of Carcinogenic Risks to Humans 71:829-864.

IARC. 1999b. Xylenes. Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide. Monographs on the Evaluation of Carcinogenic Risks to Humans 71:1189-1208.

IARC. 2000. Ethylbenzene. Some Industrial Chemicals. Monographs on the Evaluation of Carcinogenic Risks to Humans 77:227-266.

IARC. 2012. Benzene. Monographs on the Evaluation of Carcinogenic Risks to Humans. A Review of Chemical Carcinogens: Chemical Agents and Related Occupations. 100F:46 pages

IARC. 2018. Benzene. Monographs on the Evaluation of Carcinogenic Risks to Humans. 120

Infante PF, Rinsky RA, Wagoner JK, Young RJ. 1977. Leukemia in benzene workers. Lancet 2(8028):76-78.

Inoue O, Seiju K, Watanabe T, Kasahara M, Nakatsuka H, Yin S, et al. 1988. Mutual metabolic suppression between benzene and toluene in man. Int Arch Occup Envrion Health 60:15-20.

Irons RD, Lv L, Gross SA, et al., 2005. Chronic exposure to benzene results in a unique form of dysplasia. Leukemia Research 29:1371-1380.

Irons RD, Gross SA, Le A, et al. 2010. Integrating WHO 2001-2008 criteria for the diagnosis of myelodysplastic syndrome (MDS): A case-case analysis of benzene exposure. Chem. Biol. Interact. 184:30-38.

Jakobbson R, Ahlbom A, Bellander T, Lundberg I. 1993. Acute myeloid leukemia among petrol station attendants. Arch. Env. Health 48:255-259.

Lehman EJ, Hein MJ. 2006. Mortality of workers employed in shoe manufacturing: An update. Am J Ind Med 49:535-546.

Lewis, RJ, Schnatter AR, Katz AM, Thompson FS, Murray N, Jorgensen G, et al. 2000. Updated mortality among diverse operating segments of a

petroleum company. Occup Environ Med 57(9):595-604.

Linet MS, Yin SN, Gilbert ES, et al. 2015. A retrospective cohort study of cause-specific mortality and incidence of hematopoietic malignancies in Chinese benzene-exposed workers. Int J Cancer. 137:2184-97.

Lv L, Lin G, Gao X et al. 2010. Case-control study of risk factors of myelodysplastic syndromes according to World Health Organization classification in a Chinese population. Am J Hematol 86:163-169.

Lynch A, Harvey J, Aylott M, Nicholas E, Burman M, Siddiqui A, et al. 2003. Investigations into the concept of a threshold for topoisomerase inhibitor-induced clastogenicity. Mutagenesis 18(4):345-353.

Lynge E, Andersen A, Nilsson R, et al. 1997. Risk of Cancer and Exposure to Gasoline Vapors. Am J Epidemiol 145(5):449-458.

Ma X, Lim U, Park Y, Mayne ST, Wang R, Hartge P, Hollenbeck AR, Schatzkin A. Obesity, lifestyle factors, and risk of myelodysplastic syndromes in a large US cohort. 2009. Am J Epidemiol. 169:1492-9.

Medinsky MA, Schlosser PM, Bond JA. 1994. Critical issues in benzene toxicity and metabolism: the effect of interactions with other organic chemicals on risk assessment. Environ Health Perspect 9(102 Suppl):119-24.

Monson RR, Fine LJ. 1978. Cancer Mortality and Morbidity Among Rubber Workers. J Natl Cancer Inst 61(4):1047-1053.

NIOSH. 1974. Criteria for a Recommended Standard: Occupation Exposure to Benzene. National Institute of Occupational Safety and Health.

NIOSH. 1976. Revised Recommendation for an Occupation Exposure Standard for Benzene. National Institute of Occupational Safety and Health.

Nisse C, Haguenoer JM, Grandbastien B, Preudhomme C, Fontaine B, Brillet JM, et al. 2001. Occupational and environmental risk factors of the myelodysplastic syndromes in the North of France. Br J Haematol 112(4):927-935.

NTP. 2016. Report on Carcinogens, Fourteenth Edition National Toxicology Program.

Pottenger LH, Carmichael N, Banton MI, et al. 2009. ECETOC workshop on the biological significance of DNA adducts: Summary of bollow-up from an expert panel meeting. Mutation Research 678:152-157.

Poynter JN, Richardson M, Roesler M. et al. 2017. Chemical exposures and risk of acute myeloid leukemia and myelodysplastic syndromes in a

population-based study. Int J Cancer. 140:23-33.

Raabe GK, Collingwood KW, and Wong O. 1998. An Updated Mortality Study of Workers at a Petroleum Refinery in Beaumont, Texas. Am J Ind Med 33(1):61-81.

Raabe GK and Wong O. Leukemia Mortality by Cell Type in Petroleum Workers with Potential Exposure to Benzene. Environ Health Perspect 104(Suppl. 6), 1381-1392. 1996.

Rinsky RA, Smith AB, Hornung R, Filloon TG, Young RJ, Okun AH, et al. 1987. Benzene and leukemia. An epidemiologic risk assessment. N Engl J Med 316(17):1044-1050.

Rinsky RA, Hornung RW, Silver SR, Tseng CY. 2002. Benzene exposure and hematopoietic mortality: A long-term epidemiologic risk assessment. Am J Ind Med 42(6):474-480.

Rinsky RA, Young RJ, Smith AB. 1981. Leukemia in benzene workers. Am J Ind Med 2(3):217-245.

Roh J, Moon YH, Kim KY. 1987. The cytogenetic effects of benzene and toluene on bone marrow cells in rats. Yonsei. Med J 28(4):297-309.

Rushton L. 1993. Further follow up of mortality in a United Kingdom oil distribution centre cohort. British Journal of Industnial Medicine 50:561-569

Rushton L. Romaniuk H. 1997. A case-control study to investigate the risk of leukaemia associated with exposure to benzene in petroleum marketing and distribution workers in the United Kingdom. Occ Env Med.54:152-166.

Ruiz MA, Augusto LGS, Vassalo J, et al. 1994. Bone marrow morphology in patients with neutropenia due to chronic exposure to organic solvents (benzene): Early lesions. Path. Res. Pract. 190:151-154.

Samanic C, Gridley G, Chow WH, Lubin J, Hoover RN, Fraumeni JF. 2003. Obesity and cancer risk among white and black United States veterans. Cancer Cause Control 15:35-43.

Satin KP, Bailey WJ, Ross AY, Wong O. 2002. Update epidemiological study of workers at two California petroleum refineries 1950-1995. J Occup Environ Med 42(2):87-97.

Satin KP, Wong O, Yuan LA, Bailey WJ, Newton, KL, Wen C-P, et al. 1996. A 50-year mortality follow-up of a large cohort of oil refinery workers in Texas. J Occup Environ Med 38:492-506.

Schnatter RA, Katz AM, Nicolich MJ, Theriault G. 1993. A retrospective mortality study among Canadian petroleum marketing and distribution workers. Env. Health Persp. 101(Suppl. 6):85-99.

Schnatter RA, Armstrong TW, Thompson LS, et al. 1996. The relationship between low-level benzene exposure and leukemia in Canadian petroleum distribution workers. Env. Health Persp. 104(Suppl 6):1375-1379.

Schnatter RA, Glass DC, Tang G, Irons RD, Rushton L. 2012. Myelodysplastic syndrome and benzene exposure among petroleum workers: An international pooled analysis. JNCI 104:1724-37.

Schnatter RA, Glass DC, Rushton L, et al. 2012. Final Report: Pooled Analysis of Petroleum Worker Case-Control Studies. February 15, 2012

Silver SR, Rinsky RA, Cooper SP, Hornung RW, Lai D. 2002. Effect of follow-up time on risk estimates: a longitudinal examination of the relative risks of leukemia and multiple myeloma in a rubber hydrochloride cohort. Am J Ind Med. 42:481-9.

Sofuni T, Hayashi M, Nohmi T, et al. 2000. Semi-quantitative evaluation of genotoxic activity of chemical substances and evidence for a biological threshold of genotoxic activity. Mutation Research 464:97-104.

Sorahan T, Kinlen LJ, Doll R. 2005. Cancer risks in a historical UK cohort of benzene exposed workers. J Occup Environ Med 62:231-236.

Sorahan T1, Mohammed N. 2016. Incidence of Myelodysplastic Syndrome in UK Petroleum Distribution and Oil Refinery Workers, 1995-2011. Int J Environ Res Public Health. 13:474.

Straif K, Weiland SK, Werner B, Chambless L, Mundt KA, Keil U. 1998. Workplace risk factors for cancer in the German rubber industry: part 2. Mortality from non-respiratory cancers. Occup Environ Med 55:325-332.

Straube (2010) Comment on: Implications of latency period between benzene exposure and development of leukemia-A synopsis of literature, Chemico-Biological Interactions 186:248-249

Strom SS, Oum R, Elhor Gbito KY, Garcia-Manero G, Yamamura Y. 2012. De novo acute myeloid leukemia risk factors: a Texas case-control study. Cancer. 118:4589-96.

Strom SS, Gu Y, Gruschkus SK, Pierce SA, Estey EH. 2005. Risk factors of myelodysplastic syndromes: a case-control study. Leukemia 19(11):1912-1918.

Svensson BG, Nise G, Englander V, Attewell R, Skerfving S, Moller T. 1990. Deaths and tumours among rotogravure printers exposed to toluene. Br J Ind Med 47(6):372-379.

Talibov M, Lehtinen-Jacks S, Martinsen JI, Kjærheim K, Lynge E, Sparén P, Tryggvadottir L, Weiderpass E, Kauppinen T, Kyyrönen P, Pukkala E. Scand . 2014. Occupational exposure to solvents and acute myeloid leukemia: a population-based, case–control study in four Nordic countries. J Work Environ Health 2014:511-517

Tice RR, Vogt TF, Costa DL. 1982. Cytogenetic effects of inhaled benzene in murine bone marrow. Genotoxic Effects of Airborne Agents 257-275. New York, Plenum Press.

Triebig G. 2010a. Implications of latency period between benzene exposure and development of leukemia—A synopsis of literature. Chemico-Biological Interactions 184:26–29.

Triebig G. 2010b. Response to the Letter to the Editor. Implications of latency period between benzene exposure and development of leukemia—A synopsis of literature. Chemico-Biological Interactions 186:247.

Tsai S, Ahmed F, Wendt JK, Foster D, Donnelly RP, Strawmyer TR. 2007. A 56-year mortality follow-up of Texas petroleum refinery and chemical employees, 1948-2003. J Occup Environ Med 49(5):557-567.

Tunek A, Hogstedt B, Olofsson T. 1982. Mechanism of benzene toxicity. Effects of benzene and benzene metabolites on bone marrow cellularity, number of granulopoietic stem cells and frequency of micronuclei in mice. Chem Biol Interact 39(2):129-138.

U.S. EPA. 2005. Risk Assessment Forum. Guidelines for Carcinogen Risk Assessment. U.S. Environmental Protection Agency EPA/630/P-03/001F. Washington, DC.

U.S EPA. 2019. Integrated Risk Information System. https://www.epa.gov/iris

U.S. Public Health Service. 1964. Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service. Washington, DC

U.S. Public Health Service. 2004. Surgeon General's Report. Chapter 2, Cancer. U.S Public Health Service. pp 35-360

Walker JT. Bloom TF, Stern FB, Okun AH, Fingerhut, MA, Halperin, WE. 1993. Mortality of workers employed in shoe manufacturing. Scand J Work Environ Health 19:89-95.

West RR, Stafford DA, Farrow A, et al. 1995. Occupational and environmental exposures and myelodysplasia: A case-control study. Leukemia Research 19:127-139

Wetmore BA, Struve MF, Gao P, et al. 2008. Genotoxicity of intermittent co-exposure to benzene and toluene in male CD-1 mice. Chemico-Biological Interactions 173:166-178.

WHO. 2001. Pathology and Genetics: Tumors of the Haematopoietic and Lymphoid Tissues. World Health Organization. Jaffe ES, et al., Editors. IARC Press.

WHO. 2008. WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues. World Health Organization Eds. Swerdlow SH, Campo E, Harris NL et al. IARC Press.

WHO. 2017. WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues. World Health Organization Eds. Swerdlow SH, Campo E, Harris NL et al. IARC Press.

Whysner J. 2000. Benzene-induced genotoxicity. J Toxicol Environ Health 61(5-6):347-351.

Whysner J, Reddy MV, Ross PM, Mohan M, Lax EA. 2004. Genotoxicity of benzene and its metabolites. Mutation Research 566(2):99-130.

Wilcosky TC, Marshall EG, Tyroler HA. 1984. Cancer mortality and solvent exposures in the rubber industry. Am Ind Hyg Assoc J 45(12):809-811.

Williams GM, Iatropoulos MJ, Jeffrey, AM. 2004. Thresholds for the effects of 2-acetylaminofluorene in rat liver. Toxicologic Pathology 32(Suppl. 2):85-91, 2004.

Wong O. 1987. An industry wide mortality study of chemical workers occupationally exposed to benzene. I. General results. Br J Ind Med 44(6):365-381.

Wong O, Harris F, Smith TJ. 1993. Health effects of gasoline exposure. II. Mortality patterns of distribution workers in the United States. Env. Health Persp. 101 (Suppl. 6):63-76.

Wong O. 1995. Risk of acute myeloid leukaemia and multiple myeloma in workers exposed to benzene. Occup. Env. Med. 52:180-384.

Wong O, Raabe GK. 1995. Cell-Type-Specific Leukemia Analyses in a Combined Cohort of More Than 208,000 Petroleum Workers in the United States and the United Kingdom, 1937-1989. Regul Toxicol Pharm 21(2):307-321.

Wong O, Harris F, Rosamilia K, Raabe GK. 2001. An Updated Mortality Study of Workers at a Petroleum Refinery in Beaumont, Texas 1945 to 1996. J Occup Environ Med 43(4):384-401.

Wong O, Harris F, Armstrong TW, Hua F. 2010. A hospital-based case-control study of acute myeloid leukemia in Shanghai: Analysis of environmental and occupational risk factors by subtypes of the WHO classification. Chem-Bio Interact 184:112-128.

Yin S-N, Hayes RB, Linet MS, et al. 1996. A Cohort Study of Cancer Among Benzene-Exposed Workers in China: Overall Results. American Journal of Industrial Medicine. 29:227-235 (1996)

Yin SN, Li GL, Tain FD, Fu ZI, Jin C, Chen YJ, et al. 1989. A Retrospective Cohort Study of Leukemia and Other Cancers in Benzene Workers. Environ Health Persp 82:207-213.

April, 2019

JOHN WHYSNER, M.D., Ph.D., D.A.B.T.

109 Victoria Street
Santa Fe, New Mexico 87505
914-582-0432
whysner@gmail.com

WORK EXPERIENCE

1982 - 2019	*Vice President*, Washington Occupational Health Associates, Inc. Washington, DC
2003 - 2014	*Associate Clinical Professor of Environmental Health Sciences*, Mailman School of Public Health, Columbia University, NY, NY
1989 - 2002	*Head*, Toxicology and Risk Assessment Program *Chief,* Division of Pathology and Toxicology American Health Foundation, Valhalla, NY
1974 - 1982	*President*, Medical Research Applications, Inc., Washington, DC
1973 - 1974	*Director of Biomedical Research*, Special Action Office for Drug Abuse Prevention, Executive Office of the President, U.S.A.
1971 - 1973	*Research Associate*, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland
1960 - 1964	*Research Associate*, Department of Pharmacology, University of Southern California School of Medicine

EDUCATION

1970 - 1971	Pediatric Resident Bronx Municipal Hospital (Albert Einstein College of Medicine), New York, New York
1964 - 1970	University of Southern California School of Medicine, Combined M.D. - Ph.D. Program in Biochemistry, Los Angeles, California
1961 - 1964	Johns Hopkins University Undergraduate Biology Program, Baltimore, Maryland

PROFESSIONAL MEMBERSHIPS, CONSULTING AND AFFILIATIONS

Diplomat, American Board of Toxicology (Board Certification)

International Agency for Research on Cancer (IARC) Working Groups

Society of Toxicology

American College of Occupational and Environmental Medicine

National Institute of Environmental Health Sciences (NIEHS)

Agency for Toxic Substances and Disease Registry (ATSDR)

Environmental Protection Agency

New York Occupational Medical Association

American Association for the Advancement of Science

International Society of Regulatory Toxicology and Pharmacology

Lecturer, School of Public Health, Yale University

MEDICAL LICENSES

1972 - 2010 District of Columbia

1973 - 1976 Pennsylvania

MILITARY SERVICE

1971 - 1973 United States Public Health Service

FIELDS OF EXPERIENCE

Toxicology and Risk Assessment

Laboratory Research -- Principle investigator of mechanisms of chemical-induced cancer development, especially of brain tumor development, PCB effects in experimental animals related to type of PCBs and congener mixture, oxidative DNA damage and benzene-induced leukemia. PCB research included analysis of bioassay results for mechanism of cancer in rats used for risk assessment by the EPA. Methods employed include detection of oxidized nucleosides using electrochemical detection, DNA adduct formation by ^{32}P-postlabeling and DNA isolation and radiolabeled binding. Other research activities include supervision of animal bioassays including initiation-promotion studies, immunohistochemical analysis and pathological evaluation of brain, liver, kidney and forestomach tumors.

Cancer Hazard Identification -- Consultant to the International Agency for Cancer Research and "ORD" subgroup leader for Monographs 73 and 79, which focused on cancer mechanisms they relate to hazard identification. These working groups evaluated whether the animal studies reliably identified chemicals with potential to cause human cancer. Also the results of epidemiology studies and animal bioassays were reviewed for the chemicals evaluated.

Cancer Mechanism and Risk Assessment -- Project Leader for an international panel of experts involved in determining the impact of cancer mechanism on risk assessment. The chemicals requiring complete reviews of cancer data for use as examples include benzidine, vinyl chloride, formaldehyde, diethylnitrosamine, 2-acetylaminofluorine, 2,3,7,8,-tetrachlorodibenzodioxin (TCDD), phenobarbital, polychlorinated biphenyls (PCBs), d-limonene, saccharin, butylated hydroxyanisole, and clofibrate.

Risk Assessment -- Author of several site-specific risk assessments involving soil and sediment remediation for organic chemicals and metals including arsenic, beryllium, chromium and lead. Some of these included the development of corporate remediation standards for cleanup of soil and surfaces prior to the availability of Federal standards.

Risk Communication -- Provided risk communication regarding public concerns related to potential toxic effects of fragrances and flavors, smokestack emissions from power plants and the presence of chemicals in soils, groundwater and fish.

Health Effects of Air Pollutants -- Analysis of health effects of criteria pollutants and trace air emissions including potential carcinogenic effects. Modeled air emissions compared to levels at which health effects may be found. Qualified as an expert witness; written and oral testimony provided. Emissions involved included sulfur dioxide, particulates, nitrogen oxides, sulfuric acid mist,

carbon monoxide, lead, beryllium, fluorine, mercury, antimony, arsenic, barium, cadmium, chromium, cobalt, copper, hydrochloric acid, manganese, nickel, phosphorus, zinc, formaldehyde, phenol, polycyclic organic material (PAHs), and pyridine.

Polychlorinated Hydrocarbons -- Carcinogenic risk assessment, medical surveillance for occupational exposures to polychlorinated biphenyls, polychlorinated dibenzofurans, dioxins, tri- and tetrachloroethylene and related hydrocarbons. Health effects analysis of PCBs and potential health effects related to congener mix for the Hudson River, Mud River, Fox River and New Bedford Harbor. Author of a complete review of the human literature, several risk assessments and the development of clean-up standards for PCBs including the Paoli and Wilmington rail yards.

Lead Exposure Effects -- Author of several monographs contracted by the Departments of Health and Human Services, Housing and Urban Development and the National Bureau of Standards concerning prevention, treatment and assessment of lead exposure effects. Designed several studies including the New York City Health Department study of lead poisoning sources. Testing, evaluation, remediation and risk communication for lead in drinking water.

Petroleum Products -- Risk assessment, medical evaluation, worker protection evaluation for petroleum-derived solvents and oils. Author of a comprehensive review of mechanisms of benzene-induced leukemogenesis, especially the possible role of genotoxicity and DNA reactivity and topoisomerase inhibition.

Occupational Medicine

Hazard Communication -- Design and implementation of hazard communication programs. Development of Material Safety Data Sheets (MSDSs), emergency response procedures for chemical exposures, and special presentations on hazardous chemicals.

Medical Surveillance -- Design, implementation, and evaluation of medical programs in the hazardous waste industry, manufacturing, and transportation.

Pharmaceutical Development

Investigations of New Drugs -- Design, supervision analysis and authorship of several clinical toxicology studies in the field of opiate analgesics and antagonists. One of these studies examined the efficacy and toxicity of a long-acting opiate in 4,000 patients for two years.

New Drug Application -- Author of one complete NDA including analysis of three Phase II and III studies, complete literature review and medical monitors summary.

Pharmacology and Biochemistry

Steroid and Drug Metabolism -- Investigations of the cytochrome P-450 oxidase system for hormone and drug metabolism.

Heavy Metal Enzyme Chemistry -- Author of several publications regarding electron spin resonance spectroscopy and enzymatic assays.

Neurological Pharmacology -- Neurochemical and electrophysiological techniques applied to receptor identification, binding and isolation. Radioisotopic coupling with specific receptor sites.

PUBLICATIONS

Huang, S. X. L., Jaurand, M-C., Kamp D. W., Whysner, J., Hei, T. K. Role of mutagenicity in asbestos fiber-induced carcinogenicity and other diseases. Journal of Toxicology and Environmental Health, Part B, 14:1-67 (2011).

Gulati, A.P, Yang, Y-M, Harter, D., Mukhopadhyay, A., Aggarwal, B.A., Benzil, D.L., Whysner, J., Albino, A.P., Murali R., Jhanway-Uniyal, M. Mutant Human Tumor Suppressor p53 Modulates the Activation of Mitogen-Activated Protein Kinase and Nuclear Factor-kB, But Not C-Jun N-Terminal Kinase and Activated Protein-1, Molecular Carcinogenesis, 45:26-37 (2006).

Whysner, J. Quantitative Cancer Risk Assessment, In: *Cancer Risk Assessment*, P.J. Shields (Ed), Taylor & Francis Group. LLC (2005)

Whysner, J., Reddy, M.V., Ross , P.M., Mohan, M., Lax, E.A. Genotoxicity of Benzene and Its Metabolites, Mutation Research, 566:99-130 (2004).

Kumar, S., Lin, J-M, Whysner, J., Sikka, H.C., Amin, S. Mutagenicity of Benzo[b]phenanthro[2,3-d]thiophene (BPT) and Its Metabolites in TA100 and Base-Specific Tester Strains (TA7001-TA7006) of *Salmonella Typhimurium*: Evidence of Multiple Pathways for the Bioactivation of BPT. Mutation Research, 545:11-21 (2004).

Whysner, J. and Chase, K.H., Risk Assessment, In: *A Practical Approach to Occupational and Environmental Medicine*, R.J. McCunney, (Ed.), Third Edition, 2003.

Groves, F.D., Issaq, H., Fox, S., Jeffrey, A.M., Whysner, J., Zhang, L., You, W.C., Fraumeni, J.F. Jr. N-nitroso compounds and mutagens in Chinese fermented (sour) pancakes, J. AOAC Int., 85: 1052-6 (2002).

Whysner, J., Identification and Classification of Carcinogens, In *Carcinogens in the*

Workplace, J. Whysner and P.J. Shields (Eds.), *Clinics in Occupational and Environmental Medicine*, W. B. Saunders Company Science, Philadelphia, 2002.

Yang, Y-M., Conaway, C.C., Chiao, J.W., Wang, C-X., Amin, S. Whysner, J., Dai, W., Reinhardt, J., Chung, F-L. Inhibition of Benzo(a)pyrene-Induced Lung Tumorigenesis in A/J mice by Dietary *N*-acetylcysteine Conjugates of Benzyl and Phenethyl Isothiocyanates During the Post-initiation Phase is Associated with Activation of MAP Kinases and P53 activity and Induction of Apoptosis, *Cancer Research*, 62:2-7, 2002.

Brown, J.F., Fish, K.M., Mayes, B.A., Silkworth, J.B., Hamilton, S.B., and Whysner, J. PCB Effects on Epigenetic Mechanisms. *PCBs: Recent Advances in Environmental Toxicology and Health Effects*, Eds. L.W. Robertson and L.G. Hansen, University of Kentucky Press, Lexington, KY, pp. 329-336, 2001.

Whysner, J., Wang C-X. Hepatocellular Iron Accumulation and Increased Proliferation in Polychlorinated Biphenyl-Exposed Sprague-Dawley Rats and the Development of Hepatocarcinogenesis. *Toxicological Sciences*, 62:36-45, 2001.

Whysner, J., Benzene-Induced Genotoxicity. *Journal of Toxicology and Environmental Health*, Part A, 61:347-351, 2000.

Whysner, J., Mohan, M. Perineal Application of Talc and Cornstarch Powders: Evaluation of Ovarian Cancer Risk. *American Journal of Obstetrics and Gynecology*, 182:720-724, 2000.

Williams, G.M., Iatropoulos, M.J., Whysner, J. Safety Assessment of Butylated Hydroxyanisole and Butylated Hydroxytoluene as Antioxidant Food Additives. *Food and Chemical Toxicology*, 37:1027-38, 1999.

Ross, P.M., Whysner, J., Covello, V.T., Kuschner, M., Rifkind, A.B., Sedler, M.J., Trichopoulos, D., and Williams, G.M.. Olfaction and Symptoms in the Multiple Chemical Sensitivities Syndrome. *Preventive Medicine*, 28: 467-480, 1999.

Whysner, J., Montandon, F., McClain, R.M., Downing, J., Verna, L.K., Steward, R.E., 3rd and Williams, G.M. Absence of DNA Adduct Formation by Phenobarbital, Polychlorinated Biphenyls and Chlordane in Mouse Liver Using the ^{32}P-Postlabeling Assay. *Toxicology and Applied Pharmacology*, 148:14-23, 1998.

Whysner, J., Steward, R.E., 3rd, Chen, D., Conaway, C.C., Verna, L.K., Richie, J.P., Jr., Ali, N., and Williams, G.M. Formation of 8-Oxodeoxyguanosine in Brain DNA of Rats Exposed to Acrylonitrile. *Archives of Toxicology*, 72:429-438, 1998.

Whysner, J., Conaway, C.C., Verna, L.K., Ross, P.M., Williams, G.M. Evaluation of Possible Genotoxic Mechanisms for Acrylonitrile Tumorigenicity. *Regulatory*

Toxicology & Pharmacology, 27:217-239, 1998.

Whysner J. Epidemiology of Silicone Breast Implants. *Annals of Internal Medicine*. 126:667, 1998.

Hard, G.C., Whysner, J., English, J.C., Zang, E. & Williams, G.M. Relationship of Hydroquinone-Associated Rat Renal Tumors with Spontaneous Chronic Progressive Nephropathy. *Toxicologic Pathology*. 25:132-143, 1997.

Whysner, J., Conaway, C.C., Verna, L. & Williams, G.M. Vinyl Chloride Mechanistic Data and Risk Assessment: DNA Reactivity and Cross-Species Quantitative Risk Extrapolation. *Pharmacology & Therapeutics*. 71:7-28, 1996.

Conaway, C.C., Whysner, J., Verna, L. & Williams, G.M. Formaldehyde Mechanistic Data and Risk Assessment: Endogenous Protection from DNA Adduct Formation *Pharmacology & Therapeutics*. 71:29-55, 1996.

Verna, L., Whysner, J. & Williams, G.M. N-Nitrosodiethylamine Mechanistic Data and Risk Assessment: Bioactivation, DNA Adduct Formation, Mutagenicity and Tumor Initiation. *Pharmacology & Therapeutics*. 71:57-81, 1996.

Verna, L., Whysner, J. & Williams, G.M. 2-Acetylaminofluorene Mechanistic Data and Risk Assessment: DNA Reactivity, Enhanced Cell Proliferation and Tumor Initiation. *Pharmacology & Therapeutics*. 71:83-105, 1996.

Whysner, J., Verna, L. & Williams, G.M. Benzidine Mechanistic Data and Risk Assessment: Species and Organ-Specific Metabolic Activation. *Pharmacology & Therapeutics*. 71:107-126, 1996.

Whysner, J. & Williams, G.M. d-Limonene Mechanistic Data and Risk Assessment: Absolute Species-Specific Cytotoxicity, Enhanced Cell Proliferation and Tumor Promotion. *Pharmacology & Therapeutics*. 71:127-136, 1996.

Whysner, J. & Williams, G.M. Butylated Hydroxyanisole Mechanistic Data and Risk Assessment: Conditional Species-Specific Cytotoxicity, Enhanced Cell Proliferation and Tumor Promotion. *Pharmacology & Therapeutics*. 71:137-151, 1996.

Whysner, J., Ross, P.M. & Williams, G.M. Phenobarbital Mechanistic Data and Risk Assessment: Enzyme Induction, Enhanced Cell Proliferation and Tumor Promotion. *Pharmacology & Therapeutics*. 71:153-191, 1996.

Whysner, J. & Wiliams, G.M. 2,3,7,8-Tetrachlorodibenzodioxin Mechanistic Data and Risk Assessment: Gene Regulation, Cytotoxicity, Enhanced Cell Proliferation and Tumor Promotion. *Pharmacology & Therapeutics*. 71:193-223, 1996.

Whysner, J. & Williams, G.M. Saccharin Mechanistic Data and Risk Assessment: Urine Composition, Enhanced Cell Proliferation and Tumor Promotion. *Pharmacology & Therapeutics.* 71:225-252, 1996.

Williams, G.M. & Whysner, J. Epigenetic Carcinogens: Evaluation and Risk Assessment. *Experimental & Toxicologic Pathology.* 48:97-103, 1996.

Whysner, J., Verna, L., English, J.C., Williams, G.M., "Analysis of studies related to tumorigenicity induced by hydroquinone", *Regulatory Toxicology and Pharmacology.* 21:158-176, 1995.

Olsen, J.H., Schulgen,G., Boice, J.D., Whysner, J., Travis, L.B., Williams, G.M., Johnson, F.B., O'D McGee, J., "Antiepileptic treatment and risk for hepatobiliary cancer and malignant lymphoma", *Cancer Research.* 55:294-297, 1995.

Williams, G.M. and Whysner, J., "Mechanistic Considerations in Risk Assessment for Tumor Promoters," In: *Growth Factors and Tumor Promotion: Implications for Risk Assessment,* R.M. McClain, T.J. Slaga, (Eds.), John Wiley & Sons, Inc., New York, pp. 369-383, 1995.

Hard, G.C. and Whysner, J., "Risk Assessment of d-Limonene: An Example of Male Rat-Specific Renal Tumorigens", *Critical Reviews in Toxicology*, 24:231-254, 1994.

Whysner, J., "The Use of Modeled Air Emission Data by the Environmental Health Physician", *Proceeding of the Second International Conference on Managing Hazardous Air Pollutants, 1993,* Electric Power Research Institute. 1994.

Whysner, J. and Chase, K.H., "Risk Assessment", In: *A Practical Approach to Occupational and Environmental Medicine,* R.J. McCunney, (Ed.), Second Edition, pp. 376-382, 1994.

Whysner, J., Kuschner, M., Covello, V.T., Rifkind, A.B., Rozman, K.K., Trichopoulos, D., Williams, G.M., "Asbestos in the Air of Public Buildings: A Public Health Risk?", *Preventive Medicine,* 23:119-125, 1994.

Whysner, J., Wang, C., Zang, E., Iatropoulos, M.J. and Williams, G.M., Dose-Response of Promotion by Butylated Hydroxyanisole in Chemically Initiated Tumors of the Rodent Forestomach", *Food and Chemical Toxicology,* 32:215-222, 1994.

Whysner, J., "Mechanism-Based Cancer Risk Assessment of Butylated Hydroxyanisole, *Toxicology and Industrial Health,* 9(1-2):283-293, 1993.

Williams, G.M., Verna, L.K. and Whysner, J., "Mechanisms of Chemical Carcinogenesis: Application to Safety Assessment of Pesticides", In: *Brighton Crop Protection Conference--Pests and Diseases,* BCPC Publications, Surrey, England, 1:135-142.

1992.

Whysner, J.A. and Williams G.M., "International Cancer Risk Assessment: The Impact of Biologic Mechanism," *Regulatory Toxicology and Pharmacology*, 15, 41-50, 1992.

Shields, P.G., Whysner, J.A., Chase, K.C., "Polychlorinated Biphenyls and Other Polyhalogenated Aromatic Hydrocarbons", In: *Hazardous Materials Toxicology*, J. Sullivan, (Ed.), Williams & Wilkins, Baltimore, 1992.

Chase, K.C. and Whysner, J.A., "Risk Assessment in Occupational Health" in McCunney, R., (Ed.), In: *Handbook of Occupational Medicine*, pp. 324-332, 1988.

Blaine, J., Renault P., Thomas D.B. and Whysner, J.A., "Clinical Status of Methadyl Acetate (LAAM)", Research Development in Drug and Alcohol Use, R.B. Millman, P. Cushman and J.H. Lowinson, (Eds.), *Annals of the New York Academy of Sciences*, 362:101-115, 1981.

Blaine, J., Thomas, D.B., Barnett, G., Whysner, J.A. and Renault, P.G.,"Levo-Alpha-Acetyl Methadol: Clinical Utility and Pharmaceutical Development", In: *Substance Abuse: Clinical Problem and Perspectives*, J.H. Lowinson and P. Ruix, (Eds.), pp. 360-388, 1981.

Blaine JD, Renault PR, Thomas DB, Whysner JA. 1981. Clinical status of methadyl acetate (LAAM). Ann N Y Acad Sci. 362:101-15.

Whysner JA, Thomas DB, Ling W, Charuvastra C. 1979. On the relative efficacy of LAAM and methadone. NIDA Res Monogr. 27:429-33.

Whysner, J.A., "Phase III Clinical Study of Levo-Alpha-Acetyl Methadol", In: *Critical Concerns in the Field of Drug Abuse*, J.H. Lowinson, B.J. Primm, S.D. Coletti, (Eds.), pp. 1321-1326, 1978.

Blaine, J., Renault, P., Levine, G.L. and Whysner, J.A., "Clinical Use of LAAM". Recent Developments in Chemotherapy of Narcotic Addiction, B. Kissin, J.H. Lowinson, R.B. Millman, (Eds.), *Annals of the New York Academy of Sciences*, 311:214-231, 1978.

Whysner, J.A. and Levine, G.L., "Phase III Clinical Study of LAAM: Report of Current Status and Analysis of Early Terminations" In: *The International Challenge of Drug Abuse*, NIDA Monograph 19:277-290, 1978.

Whysner, J.A., "Phase III Clinical Study of Levo-Alpha-Acetylmethadol." In Rx: 3X/Week LAAM Alternative to Methadone. Ed. J. Blain and P. Renault. NIDA Research Monograph 8:109-111, 1976.

Whysner, J.A., Ramseyer, J. and Harding B.W., "Substrate Induced Changes in Visible Absorption and Electron Spin Resonance Properties of Adrenal Cortex Mitochondrial P450", *The Journal of Biological Chemistry*, 245:5441-5449, 1970.

Harding, B.W., Bell, J.J., Wilson, L.D. and Whysner, J.A., "Biosynthesis of Adrenocorticosteroids: Energy Metabolism and the Hydroxylases", In: *Advances in Enzyme Regulation*, G. Weber, (Ed.), 7:237-257, 1969.

Whysner, J.A., Ramseyer, J., Kazmi, G.M. and Harding, B.W., "Substrate Induced Spin State Changes in Cytochrome P450", *Biochemical and Biophysical Research Communication*, 36:795-801, 1969.

Harding, B.W., Oldham, S.B., Whysner, J.A. and Wilson, L.D., "Corticosteroid Biosynthesis by Adrenal Mitochondria", In: *Biogenesis and Action of Steroid Hormones*, Dorfman, R.I., et. al., (Eds.), pp. 140-203, 1968.

Whysner, J.A. and Harding, B.W., "Substrate Interaction with Cytochrome P450 of Adrenal Cortical Submitochondrial Particles", *Biochemical and Biophysical Research Communication*, 32:921-927, 1968.

Whysner, J.A. and Saunders, P.R., "Purification of the Lethal Fraction of the Venom of the Marine Snail Conus Californicus", *Toxicon*, 4:177-181, 1966.

Whysner, J.A. and Saunders, P.R., "Studies on the Venom of the Marine Snail Conus Californicus", *Toxicon*, 1:113-122, 1963.

Trial Testimony Previous Four Years and Fee Schedule: (Revised 5/30/2019)

Ralph Girardi vs. Berryman Products, Inc. et al. United States District Court, District of Massachusetts. Case No. 1:14-CV-12853. (Deposition March 11, 2016)

David Dominguez and Amanda Dominguez vs. CYTEC Industries, Inc. et al. Superior Court of the State of California County of Los Angeles, Central District. Case No. BC 533123. (Deposition July 21, 2016)

Oliver Bouteiller, et al. v. Service Auto Parts, Inc., et al. Superior Court, J.D. Waterbury at Waterbury. Docket No.: (X10) UWY-CV-14-6027407-S. (Deposition September 16, 2016)

Regina Eaves, et al., vs. Ashland, Inc., et al. Superior Court of California, County of Contra Costa. Case No.: MSC16-00815. (Deposition November 14, 2018; Trial March 21, 2019)

Daryl Deaton and Kristi Patterson individually and as Personal Representatives of the Estate of Walter Deaton vs. United States Steel Corporation, et al. United States District Court for the District of South Carolina Charleston Division. Case No.: 2:19-cv-00204-RMG. (Deposition May 20, 2019)

Jimmy H. Thomas and Sonya Thomas vs. AKZO Nobel Coatings, Inc. Superior Court of the State of California for the County of Alameda. Case No. RG17882514. (Deposition May 30, 2019)

Hourly rate for John Whysner MD, PhD, DABT is $650 payable to Washington Occupational Health Associates, Inc. Tax ID# 52-1195818

EXHIBIT B

Ethan A. Natelson, M.D., F.A.C.P.
Board Certified in Internal Medicine and Hematology

8707 Wateka Drive
Houston, Texas 77074-4015

Telephone: (713) 441-5154
Fax: (713) 793-7065
E-mail: natelson@pipeline.com

July 15, 2019

Mr. Brett J. Young
Norton Rose Fulbright US LLP
1301 McKinney, Suite 5100
Houston, Texas 77010-3095

Re: *Mary Major (Elwyn Webb, deceased) v. SFPP, LP et al.*, No. CV 2018-003217, Superior Court of Arizona

Dear Mr. Young,

Thank you for allowing me to review the medical records that you have provided concerning the above action. As you are aware, I am a Board-certified hematologist in clinical practice at Houston Methodist Hospital (HMH), in Houston, Texas, where I have been Director of the Transitional Residency Program. I am currently one of four designated key faculty members facilitating our Internal Medicine Residency Program. I also Chair one of our two Internal Review Boards (IRBs) which must review, discuss, approve and monitor research on human subjects involving HMH, where we currently maintain approximately 1,100 active protocols. I have a faculty appointment as Professor of Clinical Medicine at Weill-Cornell Medical College which is affiliated with HMH and a similar appointment to the HMH Research Institute. I also have an adjunct Professorship at the Texas A&M Medical School and a similar clinical faculty appointment to the University of Texas Medical Branch in Galveston, Texas. These latter two institutions assign a large number of their medical students for clinical teaching on our medical services at HMH, in which I participate as an attending physician. I have previously held full academic faculty appointments to Baylor Medical School and The University of Texas Medical School at Houston (*renamed The University of Texas McGovern Medical School*). Until earlier this year I had been a long-time member of the Board of Directors of the Gulf Coast Regional Blood Center, where I chaired its Education and Research Committee, and served on its Technical Advisory committee. I am a past president of the Gulf Coast Hematology Society, and the Houston Society of Internal Medicine.

I have more than 50 years' experience in the field of clinical hematology and have been a member of the American Society of Hematology since 1971. I am familiar with

the hematological disorders (*neoplasms*) classified as leukemias such as acute myeloid leukemia (AML), the myelodysplastic syndromes (MDS) and the classical myeloproliferative neoplasms (MPN) such as polycythemia vera, primary thrombocytosis and primary myelofibrosis. I also am familiar with the more recently established World Health Organization (WHO) category of myelodysplastic/myeloproliferative neoplasms (**MDS/MPN**). This latter group includes hematological neoplasms such as atypical chronic myeloid leukemia (aCML) and chronic myelomonocytic leukemia (CMML) as well as currently unclassifiable forms of **MDS/MPN**. I am also aware of modern opinion and teaching concerning what is known as to the etiology, diagnosis, classification, clinical course and therapy of these hematological neoplasms. I continue to see patients with these and other diverse hematologic syndromes in my clinical practice.

The opinions expressed herein are my own and should not be construed in any way to also represent the opinions and/or judgments of The Methodist Hospital System or any of its associated institutions and subsidiaries. My Texas medical license is current and on file with the appropriate authorities. You have a copy of my curriculum vitae and a listing of cases in which I have given testimony. My testimony has never been excluded by any court.

This action concerns Mr. Elwyn Webb (DOB 12-23-1950, DOD 03-19-2016). The medical records indicate that he underwent a hematological examination because of anemia associated with a persistently elevated white blood cell count and weight loss. As will be discussed *vide infra*, a diagnosis of a myelodysplastic/myeloproliferative neoplasm (**MDS/MPN**) was established in Mr. Webb by analysis and reports of two bone marrow examinations, and this aggressive illness led to his death. It is alleged in this action that Mr. Webb's hematological illness was a consequence of his work as a tanker truck driver and his exposure to gasoline and diesel fuels and their benzene and other chemical content. I will briefly review Mr. Webb's medical history as it is outlined in the medical records that you have sent to me and then comment concerning his specific hematological diagnosis and its association, if any, with the claims of causation made in the pleadings.

The medical records indicate that Mr. Webb underwent bone marrow examinations on 08-24-2015 and on 01-12-2016, The first sample was analyzed at the Sonora Quest Laboratories, and the second processed by the Corepath Laboratories in San Antonio, Texas. The conclusions as to a description of the bone marrow morphology and suggested specific hematological diagnosis were similar in the interpretation of the findings on both samples by these two laboratories. The bone marrows both exhibited greatly increased cellularity with estimates of almost total marrow cellularity – 90% on the first and 98 % cellularity on the second. Normal bone marrow cellularity (*based on a visual estimate of bone marrow fat versus cellular material*) is reduced as a person ages and in a man of Mr. Webb's age generally would have been about 40 % marrow cellularity, under normal circumstances. The report on the first bone marrow specimen pending results of cytogenetic studies suggested a diagnosis of "…**a myeloproliferative process…**" and the second, with the benefit of results of associated cytogenetic testing,

informs us as to the diagnosis, under the World Health Organization (WHO) guidelines, **"The overall findings are most consistent a myelodysplastic/myeloproliferative neoplasm, unclassifiable"**.

The peripheral blood count values at the second marrow included a total leukocyte count of 20,000/cu mm, a hemoglobin concentration of 7.3 gm/dl and a platelet count of 80,000/cu mm. Some nucleated erythrocytes (*normoblasts*) were present on examination of the peripheral blood film. There were 5 % myeloblasts present in both bone marrows and in the second specimen, 2 % blasts in the peripheral blood. Generally, 20 % blast forms in either the peripheral blood or bone marrow signify acute myeloid leukemia (AML). Comments on the bone marrow appearance by the hematopathologists included **"...complete trilineage hematopoiesis..."** and **"Megakaryocytes are mildly increased in numbers and are small and hypo lobulated"**. Atypical CML (aCML), a form of **MDS/MPN** was mentioned as a possible diagnosis but it was noted that, **"...the mild degree of dysplasia makes this less likely"**. There was no marrow fibrosis described. The cytogenetic studies performed did not demonstrate any specific aberrations. The JAK2 study gave negative results. The unusual morphologic finding of cytoplasmic vacuoles was noted in the bone marrow films and present in only the erythroid precursors and not the myeloid precursors, in both studies.

Cytoplasmic vacuoles are not present in normal bone marrow cells and, indeed, are uncommon observations and are seen most prominently in alcohol toxicity, copper deficiency, and with certain antibiotic toxicity - classically after use of chloramphenicol. With reference to the claims in this action, pathologists at the University of Texas MD Anderson Cancer Center point out concerning the presence of vacuoles in bone marrow cells, **"...it is noted that myeloid lineage vacuolation is not a recognized finding in MDS (*myelodysplastic syndrome*)"** (1).

Based upon his self-reports from medical records, Mr. Webb smoked at least from 1982-2008. He had been treated for cervical spine radiculopathy. He had undergone surgery on his left knee and an appendectomy and inguinal hernia repair. He also had undergone repair of an aortic abdominal aneurysm (*such aneurysms* are *a risk factor present among heavy smokers*). He had no family history of hematological disease. Mr. Webb was very weak during the course of this illness and then developed a bowel infection. For a time, he was in a skilled nursing facility and ultimately transferred to hospice care, where he died.

In discussing the microscopic appearance of a bone marrow, it is important to note that when hematologists or hematopathologists apply the descriptive terms dyspoiesis, dysplasia, myelodysplasia or dysmyelopoiesis to describe abnormal morphology of peripheral blood and bone marrow hematopoietic cells, this observation is not synonymous with the presence of a myelodysplastic syndrome (MDS). The terms relating to dysplasia may be purely descriptive and not specific for any disease classification. For example, from the chief WHO hematopathologist, Dr. Vardiman, **"Morphologic dysplasia is not specific for MDS...Inter-observer agreement among**

morphologists for recognition of dyserythropoiesis is notoriously poor, and dyserythropoiesis is the most common form of secondary dysplasia…features such as multinuclearity, irregular nuclear membranes and "megaloblastoid" changes have been observed in up to 10 % of erythroid precursors in bone marrow specimens of normal individuals" (2).

This problem in comparative classification of bone marrow disorders by morphology alone, among different observers, and even under the current WHO guidelines, is further emphasized by a recent report (3). Here, the presence or absence of morphologic cellular dyspoiesis was judged among bone marrow aspirates from 120 healthy potential bone marrow cell donors with normal blood counts analyzed without provision of clinical information, individually, by four blinded, experienced hematologic morphologists. These experts claimed more than 10 % of the marrow cells as dyspoietic among one cell lineage in 37 % of the bone marrow films studied, and as dyspoietic in 10 % of the bone marrow cells among two cell lineages in 31 % of these individuals, and, as dyspoietic among 10 % of the cells reviewed in all three (*red cell, white cell and , megakaryocytes, platelet progenitor cells or multi-lineage dysplasia)* precursor cellular lineages in 6.5 % of these specimens-all obtained from perfectly normal and healthy potential bone marrow donors with normal blood counts (3). Not surprisingly, the most recent edition of the updated 4th Edition of the WHO fascicle published in 2017, continues to slowly de-emphasize use of dysplasia for classification systems in favor of specific genetic aberrations, including specific molecular chromosome aberrations, but has not yet discarded its use (4).

And, as to causation in the WHO **MDS/MPN** category of illnesses, of importance in this action, we may read, "**The three best-defined entities within the MDS/MPN group are: chronic myelomonocytic leukemia (CMML), atypical chronic myeloid leukemia (aCML) and juvenile myelomonocytic leukemia (JMML)…The etiology of MDS/MPN is not known**" (4). Similarly, in a recent report from The University of Texas MD Anderson Cancer Center, "**The etiology of MDS/MPN is not known…**" (5). And, from a 2016 review, "**The underlying pathogenesis responsible for this group of neoplasms remain unclear as does the molecular convergency point that biologically defines the MDS/MPN category**" (6). Important in this legal action, **none** of the **MDS/MPN** disorders are proven to be benzene-related at any cumulative dose exposure level. While smoking is strongly associated with MDS, there is no proven association of smoking with the **MDS/MPN** group of neoplasms despite the significant amount of benzene in cigarette smoke.

Mr. Webb initially had some morphologic cellular dysplasia described in his bone marrow. Why does he not then have MDS, rather than **MDS/MPN**? With regard to the WHO **MDS/MPN** category we read, "**MDS/MPNs are characterized by combined 'cytopenias' and 'cytoses'. The bone marrow (BM) is usually hypercellular because of both ineffective (MDS-like) and effective (MPN-like) hematopoiesis…The clinical presentation may be more 'MDS-like' (e.g., cytopenias with dysplasia), 'MPN-like' (e.g., cytoses with organomegaly), or anywhere in between…The documentation of**

dysplasia in 1 or more HP (*hematopoietic*) lineage is a major criterion in establishing a diagnosis of MDS/MPN…" (7). Megakaryocytic dysplasia includes, **"Abnormal nuclear lobulation…Megakaryocytic clustering in core biopsy...Of note, mild to moderate myelofibrosis is a frequent finding in MDS/MPNs at the time of diagnosis…The BM is typically hypercellular"** (7). Megakaryocytic dysplasia was described in the two bone marrow examinations in Mr. Webb.

Let us now turn to what the bone marrow in Mr. Webb would have been expected to show if he had benzene-induced MDS. Although this is a very rare entity in the modern era in developed countries, we have considerable information on this subject. Hayes and associates reported on a large group of 78,000 benzene-exposed workers in China (8-9). Further information on this cohort indicated, **"Overall, findings for benzene-exposed workers with myelodysplastic syndromes included bone marrow hypocellularity and marked dyserythropoietic features"** (8). By contrast, Mr. Webb had an extremely hypercellular bone marrow, just the opposite of hypocellularity. Additionally, aside from cellular vacuolization, he had minimal dysplastic changes in the marrow.

And, from a study of factory workers in Brazil, exposed for a long period of time to benzene through an industrial accident involving a chronically leaking pipe, and found to have abnormal blood counts, the authors describe findings in 33 of these individuals who underwent bone marrow examination. We are told, "**in 152 employees…who presented with neutropenia, due to chronic exposure to benzene…all patients were removed from risk areas because of the hematological abnormalities…BM morphology was characterized by a hypocellular hemopoiesis (82%). Decrease of the granulocytic precursors (86%) was the most outstanding feature…eosinophilia in BM was observed in 71%...cell atypias and stromal changes (necrosis, increase in reticulin fibres) were frequent**" (10). Again, hypocellularity and reduced granulocytes were the outstanding features in these benzene-damaged bone marrows. By contrast, Mr. Webb had an increase in bone marrow cellularity and in granulocyte precursors and a markedly elevated white blood cell count.

And, in Dr. Irons' extensive studies of serial bone marrow examinations in heavily benzene-exposed subjects in China, we read, "**We describe a novel form of bone marrow dysplasia in 23 workers exposed to high concentrations of benzene. Distinguishing features…include dyserythropoiesis, eosinophilic dysplasia…and bone marrow hypoplasia…**" (11-12). Again, features totally unlike those findings identified in Mr. Webb who did not have bone marrow hypoplasia. In another study by Dr. Irons group, he compared the bone marrow findings in benzene-induced MDS, with those features in a large number of individuals with *de novo* MDS, and commented, "**…29 BZ highly exposed cases…were… compared to 569 cases which were determined to have no BZ exposure…Features that frequently characterized BZ signal cases included marked erythroid dysplasia in the bone marrow, no evidence of increased ring sideroblasts…a tendency toward low-to-normal BM cellularity, and a striking increase in multilineage dysplasia with abnormal eosinophils (MDS-Eo), hematophagocytosis and stromal degeneration**" (12). Hematophagocytosis is a

term describing ingestion of blood cells by overactive, large histocytic bone marrow cells which are seen by routine stains and iron stains of the bone marrow (13), As before, these findings have nothing in common with the bone marrow features described in Mr. Webb.

Therapy-induced or secondary MDS, regardless of what particular mutagenic chemical initiated it, is typically an aggressive illness commonly associated with easily-demonstrable chromosome aberrations and a poor survival (14-15). Thus, we learn, "**The majority of our t-MDS cases (88 %) had 1 or more cytogenetic abnormalities, comparable to that observed in other series of t-AML/MDS…Overall, abnormalities of chromosome 5 and /or 7 were most commonly observed…The results of our study indicate that morphologic subclassification of t-MDS is not clinically useful for risk stratification and that t-MDS as a group has an ominous outcome, similar to that of t-AML…The median survival differed by only 2 months between patients with t-MDS (median survival time, 8.5 months) and patients with t-AML (median survival time 6.5 months)**" (14).

Latency from alleged chemical or therapeutic radiation exposure to onset of hematological disease is an important concept in determining the etiology of all secondary hematologic neoplasms. With careful study of such latency periods, it has been repeatedly demonstrated that there is a characteristic window of opportunity to develop secondary MDS/AML following a toxic exposure to bone marrow cells that is generally described as around 2-15 years (17-25). Additionally, depending upon the mechanism of action of the specific chemical administered (*for example, topoisomerase II inhibitor vs. alkylating agent*), or radiation, this latency interval may be closer to the shorter or to the longer end of the 2 to 10-15-year span (16).

Not surprisingly, AML attributed to benzene exposures also appears to be associated with a defined latency period, quite similar to the modern-day chemotherapy model (7-8, 16-24). For example, the Hayes study of Chinese factory workers calculated latency from 6 months after first employment in the industry to onset of **secondary** AML/MDS (8). Nevertheless, their observations and conclusions are quite consistent with the more accurate modern-day chemotherapy data and they inform us, "**Person-years at risk were accumulated from 6 months after study entry…In our study, ANLL/MDS was linked to recent benzene exposure, and additional distant exposure did not appear to further increase risk. This finding suggests that recent benzene exposure is predictive of subsequent risk of ANLL/MDS, analogous to the short latency and wavelike increase then decrease in risk seen for several forms of radiation-induced leukemia and for chemotherapy-related AML**" (8). From another author, "**Similar to alkylating agent-associated AML, hematotoxicity and MDS often precede AML associated with benzene exposure. The latency period between exposure and disease lies between 4 months and 10 years**" (20). And, among Dr. Aksoy's large cohort of Turkish shoe workers exposed to intense benzene exposure from working with glues, in poorly ventilated areas, he pointed out the young age of his cohort (*mean, 34 years*) and commented, "**Acute myeloblastic leukemia which was seen in 14 of the 26 workers in**

our series was the most common diagnosis. The duration of exposure to benzene varied from 1-15 years with a mean of 9.7 yrs..." (18).

And from the United States Pliofilm factory cohort studies, similar observations tell us, "**The results...suggest that risk is maximal in the first 5 years since last exposure, and decreases with increasing time since last exposure...The results suggest that ensuring maximum protection of benzene workers requires assessing risk at its peak of 5-10 years since last exposure**" (19). In a summary of this type of latency from benzene exposure to disease we learn, "**Despite the fact of a relatively small number of cases, the results of several independent case-control studies came to the same conclusion: the risk of leukemia is small or almost absent 10-15 years after exposure has been stopped**" (23).

A recent update of the US Pliofilm factory cohort informs us, "**Workers in the highest** (*benzene*) **exposure categories had significantly elevated risks of ANLL** (*acute non- lymphocytic leukemia, another term for AML*) **and AML; no leukemia cases occurred in lower exposure categories. Exposure within 10 years of cancer onset appears most relevant for leukemia induction...Our results...provided further evidence of a threshold effect and relevant exposure window**" (19). These authors model the cumulative occupational benzene exposure levels among the 8 Pliofilm cohort individuals with suspected benzene-induced **secondary** AML (s-AML) as falling between 63.7 to 729 ppm years, with a mean value among the 8 individuals of 350 ppm years (19). As with other potentially leukemogenic chemicals used in clinical medicine, it is the cumulative dose of the toxin and the necessary cumulative threshold dose (CTD) achieved, not a single peak exposure that is thought related to the potential for a benzene-induced AML (16-17).

And, from the frequent plaintiff-quoted work of Dr. Glass, on the risks of benzene exposure and AML "**One implication of our findings is that exposure estimation to investigate the risks of benzene-induced leukemia can be restricted to the period up to 15 years prior to diagnosis. This should help to reduce any misclassification introduced by the uncertainty of exposure assessments carried out for jobs held in the distant past**" (24). In yet another study by Dr. Glass and her associates concerning benzene exposure and myeloproliferative diseases (MPD) and chronic myeloid leukemia (CML), and relative to Mr. Webb's myeloproliferative illness, these authors also conclude, "**No convincing association was identified between MPD or CML and low exposure to benzene**" (25). CML was originally placed in a group of classical hematological illnesses characterized by bone marrow hyperproliferative features but is now a stand- alone illness based upon its defining chromosome aberration. The designation MPD has been changed to MDN to emphasize that the myeloproliferative disorders are all considered neoplasms.

In a large, more recent study than the Hayes work, but also from China, where the potential for major benzene exposure still exists, the authors find no evidence that there is any excess of CMML, one of the **MDS/MPN** (26). This study is also done in the modern context of the **MDS/MPN** WHO category of myeloproliferative diseases that may

manifest some myelodysplastic features. There was also no increased incidence of CMML in subjects with exposures to benzene, toluene, xylene, or with exposure to gasoline, diesel fuels, and cutting oils (34). The authors further point out, pertinent to the claims in this action, that, "**We are not aware of a single regulatory agency or health organization that has recognized an association between benzene or solvent exposure and an increased risk for myeloproliferative neoplasms…Benzene exposure does not appear to be a significant predictor of CMML**" (25). Further, there is no evidence that exposure to fuels is a cause of hematologic neoplasms (26-27)

I noted that that Mr. Webb's death certificate lists an inaccurate cause of his death as MDS (myelodysplastic syndrome). Some areas of his medical chart also contain this incorrect term, to describe his hematological disease, which was documented in the two pathology reports. Unfortunately, death certificates are not scientific documents and their frequent striking inaccuracy has been discussed in the medical literature on many occasions (28-29). Mr. Webb had no documented cytogenetic aberrations identified at the detection of his hematological neoplasm, which was an established myeloproliferative disorder (**MPN** or **MDS/MPN**), not AML and certainly not MDS of a risk type likely to rapidly convert to AML. Presumably, **MPN** was the only hematologic disorder present at his death, a short time later, and not either MDS or AML. Importantly, were he to have had AML as an outcome of an underlying MDS, chromosome aberrations would likely have been present in the two marrow examinations. In their absence, his course of an alleged MDS would have been indolent, and not suddenly appearing as an aggressive rapidly progressing disorder. A sequence associated with MDS would have been reflected in his preceding blood counts over a long period of time and noted by his many physicians. This issue of MDS and prediction of its progression to AML is discussed in a recent article on MDS. Here, we are reminded, "**Cytogenetic abnormalities are the most important variable in determining prognosis…and accordingly have been included in all prognostic scoring systems…**" (30). We are provided no evidence that Mr. Webb had either MDS or a sudden onset of AML There is no reason to suspect a prior MDS based upon the known natural history of MDS, and his very specific hematopathology observations and conclusions on both bone marrow studies.

In summary, Mr. Webb's hematologic illness was thought to best represent atypical MPN or **MDS/MPN**. The two pathology report comments suggest that he would fall into the **MDS/MPN**-U category where the prognosis is very poor and therapy generally ineffective, as was his clinical course (30). There is no documentation or proof or even suggestion in the peer-reviewed scientific medical literature to base a claim that members of the **MDS/MPN** grouping of disorders are potentially benzene exposure related syndromes. Moreover, unlike MDS, which is strongly related to a history of smoking, there have been no studies suggesting this link with the **MDS/MPN**. As Dr. Marshall Lichtman, a prominent hematology educator commented concerning the likelihood of benzene or other environmental causes of any hematological malignancies, "**The infrequency of an Exogenous cause: It is likely that most blood cancers are the results of cellular misadventures in blood-forming cells or lymphocytes, aberrations of normal cell processes, such as spontaneous mutations resulting in oncogene**

formation and inadequacies of DNA repair mechanisms. Chemical: Only a small proportion of blood cancers have a cause related to exogenous or environmental factors. The only blood cancer for which there is convincing scientific evidence of an avoidable environmental cause is AML (and the closely related disease myelodysplasia), and only in a minority of cases" (32). The rarity of benzene-induced AML in developed countries in the modern era is also noted (33).

Should you require any additional information, please let me know. I would be pleased to review the bone marrow slides from this case, if they become available. I have not seen a report on what estimates of his cumulative benzene exposures were in the 10 to 15 years prior to Mr. Webb's onset of **MDS/MPN**.

Cordially yours,

Ethan A. Natelson, M.D.

REFERENCES:

1. Oo TH, Hu S. Copper deficiency –related bone marrow changes secondary to long-term total parenteral nutrition. Wiley Clinical Case Reports 2017;5:195-196.

2. Vardiman, J.W. Hematopathological concepts and controversies in the diagnosis and classification of myelodysplastic syndromes. Hematology 2006;199-204, American Society Educational Book

3. Parmentier S, et al, Dysmyelopioiesis in healthy bone marrow donors. Blood 2012;97:723-730.

4. Orazi A, et al. Myelodysplastic/myeloproliferative neioplasm, unclassifiable., pp 95-96, In WHO classification of Tumours of Haematopoietic and Lymphoid Tissues, Revised 4th Edition, Ed. Swerdlow, SH, et al, IARC Lyon, 2017.

5. Verstovsek S. Clinical update: Atypical myeloid disorders. Am J Hematol Oncol 2008;7:15-21.

6. Hall J, Foucar K. Diagnosing myelodysplastic/myeloproliferative neoplasms: laboratory testing strategies to exclude other disorders. Int J Lab Hematol 2010; doi:10:1111/j.1751-553X.2010.01250.x

7. Orazi A, et al. Myelodysplastic/myeloproliferative neoplasms, unclassifiable., pp 95-96, In WHO classification of Tumours of Haematopoietic and Lymphoid Tissues, Revised 4th Edition, Ed. Swerdlow, SH, et al, IARC Lyon, 2017

8. Hayes RB, et al. Benzene and the dose-related incidence of hematologic neoplasms in China J Natl Can Institute 1997;89:10651071

9. Linet MS, et al. Clinical features of hematopoietic malignancies and related disorders among benzene-exposed workers in China. Environ Health Perspect 1996;104:1353-1364.

10. Ruiz MA, et al. Bone marrow morphology in patients with neutropenia due to chronic exposure to organic solvents (benzene): early lesions. Path Res Pract 1994; 194:151-154.

11. Irons RD, et al. Chronic exposure to benzene results in a unique form of dysplasia. Leuk Res 2005;29:1371-1380.

12. Irons RD, et al. Integrating WHO 2001-2008 criteria for the diagnosis of myelodysplastic syndrome (MDS): A case-case analysis of benzene exposure. Chemico-Biol Interact 2010;184:30-38.

13. Natelson EA, Lynch EC, Hettig RA, Alfrey CP Jr. Histiocytic medullary reticulosis. The role of phagocytosis in pancytopenia. Arch Intern Med 1968; 122:223-2.

14. Singh ZN, Therapy-related myelodysplastic syndrome: Morphologic subclassification may not be clinically relevant. Hematopathol 2007;127:197-205.

15. Larson RA. Is Secondary Leukemia an Independent Poor Prognostic Factor in Acute Myeloid Leukemia? Best Pract & Res Clin Hematol 2007;20:29-37.

16. Natelson EA, Pyatt D. Temozolomide-Induced Myelodysplasia. Advances in Hematology 2010, doi:10.1155/2010/76402 pp1-5.

17. Natelson EA. Benzene-induced acute myeloid leukemia: A clinician's perspective. Am J Hematol 2007;82:826-830.

18. Aksoy M, et al. Leukemia in shoe-workers exposed chronically to benzene. Blood 1974;44:837-841.

19. Rhomberg L, et al. Evaluation of acute nonlymphocytic leukemia and its subtypes with updated benzene exposure and mortality estimates. A lifetable analysis of the Pliofilm Cohort. J Occup Environ Med 2016;58:414-420.

20. Morgan GJ, et al. Benzene and the hemopoietic stem cell. Chem-Biol Interact 2005;153-154:217-222.

21. Richardson DB. Temporal variation in the association between benzene and leukemia mortality. Environ Health Perspect 2008;116:370-374.

22. Silver SR, Rinsky RA, et al. Effect of follow-up time on risk estimates:A longitudinal examination of the relative risks of leukemia and multiple myeloma in a rubber hydrochloride cohort. Am J Indust Med 2002;42:481-489.

23. Triebig G. Implications of a latency period between benzene exposure and development of leukemia – a synopsis of literature. Chemico-Biol Interact 2010;84:26-29.

24. Glass DC, et al., Leukemia risk and relevant benzene exposure period- RE follow-up on risk estimates. Am J Ind Med 2002;42:481-489, and Am J Ind Med 2004;45:222-225.

25. Glass DC, et al. Risk of myeloproliferative disease and chronic myeloid leukaemia following exposure to low-level benzene in a nested case-control study of petroleum workers. Occup Environ Med 2014;71:266-274.

26. Gross SA, Irons RD, et al. A case-control study of chronic myelomonocytic leukemia (CMML) in Shanghai, China: Evaluation of risk factors for CMML, with special focus on benzene. Arch Environ Occup Health 2012;67:206-218.

27. Keenan JJ, et al. An evidence-based analysis of epidemiologic associations between lymphatic and hematopoietic cancers and occupational exposure to gasoline. Human and Experimental Toxicol 2013;32:1007-1027.

28. Hoff CH, Ratard R. Louisiana death certificate accuracy: A concern for the public's health. J Louisiana State Medical Society 2010;162:350-353.

29. Cambridge, B Cina SJ. The accuracy of death certificate completion in a suburban community. Am J Forensic Med Pathol 2010;30:232-235

30. Lee EJ, et al. The evolving field of prognostication and risk stratification in MDS: Recent developments and future directions. Blood Reviews 2016;30:1-10.

31. Smith BN, et al. Challenges in myelodysplastic/myeloproliferative neoplasms (MDS/MPN). Clinical Lymphoma, Myeloma & Leukemia 2019;19:1-8.

32. Lichtman MA. Battling the hematological malignancies. The 200 years' war. The Oncologist 2008;13:126-138.

33. Rushton L, et al. How much does benzene contribute to the overall burden of cancer due to occupation? Chemico-Biol Interact 2010;184:290-292.

CURRICULUM VITAE

Ethan Allen Natelson, M. D.

Date of preparation: July 24, 2018

A. GENERAL INFORMATION

Office Address:	6550 Fannin Street, Suite 1001 Houston, Texas 77030
Office Telephone:	713/441-5154 (Direct Line) 713/441-0006 (Clinic)
Office Fax:	713/793-7065
Home Address:	8707 Wateka Drive Houston, Texas 77074
Home Telephone:	713-771-8844
Cell Phone:	713-201-7304
Beeper:	713-768-2820
Email:	eanatelson@att.net enatelson@houstonmethodist.org
Citizenship:	USA

Optional Information:

Birth Date:	January 22, 1942
Birthplace:	King's County, New York
Marital status:	Married
Spouse's Name:	Kathy
Children's names and ages:	Marcia (45) and Laura (48)
Race/Ethnicity:	White

B. EDUCATIONAL BACKGROUND

Degree	*Institution name, city and state*	*Dates Attended*	*Year Awarded*
B.S.	Haverford College Haverford, Pennsylvania	1958-1962	1962
M.D.	Baylor College of Medicine Houston, Texas	1962-1966	1966

C. PROFESSIONAL POSITIONS AND EMPLOYMENT

Post-doctoral training including residency/fellowship

Title	*Institution Name, city and state*	*Dates*
Internship	The Methodist Hospital Houston, Texas Straight Medicine	1966 – 1967
Residency	Baylor College of Medicine Affiliated Hospitals Houston, Texas Internal Medicine	1967 – 1969
Fellowship	Baylor College of Medicine The Methodist Hospital Houston, Texas Hematology	1969 – 1970

Academic positions (teaching and research)

Title	*Institution Name, city and state*	*Dates*
Clinical Instructor	The University of Texas Medical School at San Antonio	1971-1972
Instructor	Department of Medicine Baylor College of Medicine Houston, Texas	1972 – 1973
Assistant Professor of Medicine	Baylor College of Medicine Houston, Texas	1973 – 1975
Clinical Assistant Professor of Medicine	Baylor College of Medicine Houston, Texas	1975 – 2006
Adjunct Associate Professor of Biomedical Engineering	Rice University Houston, Texas	1975 – 1980
Clinical Associate Professor	University of Texas Health Science Center at Houston Houston, Texas	1975 – 2006
Visiting Professor	Northwest Forestry University, State Education Commission, China	Aug 1997

Associate Professor Of Clinical Medicine	Weill Cornell Medical College New York, NY	2007-2012
Professor of Clinical Medicine	Weill Cornell Medical College	2012-present
Adjunct Clinical Professor of Medicine	Univ. of Texas Med. Branch Galveston	2013 – present
Professor of Clinical Medicine	Institute for Academic Medicine-HMH	2014 – present
Adjunct Professor of Clinical Medicine	Texas A&M Medical School	2016 - present

Hospital positions (attending physician, if applicable)

Title	*Institution Name, city and state*	*Date*
Attending Physician	Houston Methodist Hospital Houston, Texas	1972 – 1975
Consultant in Hematology	Veterans Administration Hospital Houston, Texas	1972-1975
Consulting Staff	The Methodist Hospital Houston, Texas	1975 – 2006
Active Staff	Christus St. Joseph Hospital Houston, Texas	1975 – 2007
Academic Chief of Medicine	Christus St. Joseph Hospital Houston, Texas	1975 – 2007
Director of Medical Education	Christus St. Joseph Hospital Houston, Texas	1988 – 1998
Director of The Transitional Residency Program	St. Joseph Hospital, Houston, Texas The Methodist Hospital, Houston, Texas	1988 - 2007 2007- 2016
Key Faculty, Internal Medicine Residency Program	Houston Methodist Hospital	2016 - Present
Active Staff	Houston Methodist Hospital Houston, TX	1/2006-Present
Promotion and Appointment Review Committee, HMH		6/20/2014-Present
Interim Director of the Methodist Hospital Houston Internal Medicine Residency Program (ACGME #1404813534)		5/8/2018 -6/14/2018

Employment (other than positions listed above)

Title	*Institution Name, city and state*	*Dates*
Military Service	Major, United States Air Force	1970 – 1972
Private Practice	Stehlin & de Ipolyi Oncology Clinic, P.A 1315 St. Joseph Parkway, Suite 1800 Houston, Texas 77002	August 1985-May 2005
Private Practice	Surgical Oncology Consultants of Houston, LLP 1315 St. Joseph Parkway, Suite 1800 Houston, Texas 77002	June 2005-May 2006

D. LICENSURE, BOARD CERTIFICATION, MALPRACTICE (if applicable)

Licensure

State	*Number*	*Date of Issue*	*Date of last registration*
Texas	D9036	Jan 1972	Exp. 05-31-18

NPI Number: 1417921032

NCI Investigator Number: 12301

DEA Number: On request

Texas DPS Number: On request

Board Certification

Name of specialty	*Board Certificate Number*	*Date of Certification*
American Board of Internal Medicine (ABMS)	033221	October 15, 1974
American Board of Internal Medicine-Hematology	033221	October 15, 1974

Malpractice Insurance

Name of Provider: The Methodist Hospital Physician Association

E. PROFESSIONAL MEMBERSHIPS *(medical and scientific societies)*

Member/officer	*Name of Organization*	*Dates held*
Member	American College of Physicians	1975 - Present
Member	American Society of Hematology	1971- Present
Board Member	**Gulf Coast Regional Blood Center** Chairman Educational & Research Committee	2002- 2018

Member	**Gulf Coast Society of Hematology** President	1972 - Present 2005
Member	Harris County Medical Society	1972- Present
Member	Houston Society of Internal Medicine President	1972-2007 2000-2002
Member	Texas Club of Internists	1986 - Present
Member	Texas Medical Association	1972 - Present

F. HONORS AND AWARDS

Name of award	*Date awarded*
Intern of the Year The Methodist Hospital Houston, Texas	1966
Best Teacher of the Year Christus St. Joseph Hospital Houston, Texas	1984, 1985, 1991, 1995, 1997
Exemplary Leadership Award Texas Agricultural Extension Service	1997, 2009
Distinguished Faculty Award, Houston Methodist Hospital	2010, 2016

G. INSTITUTIONAL/HOSPITAL AFFILIATION

Primary Hospital Affiliation: Houston Methodist Hospital (HMH)

H. EMPLOYMENT STATUS

Name of Employer(s): Houston Methodist Hospital Physician Organization

Employment Status: Full-time salaried at Cornell-affiliated hospital

I. CURRENT AND PAST INSTITUTIONAL RESPONSIBILITIES AND PERCENT EFFORT

Teaching ***Dates***

In 1975, I joined the faculty at UTMSH with a full academic appointment. I came there from Baylor Medical School, where I also had an academic appointment and was a member of the hematology section. I was stationed at St. Joseph Hospital and in charge of internal medicine training for the Transitional Internship at St. Joseph Hospital and in charge of 12 UTMSH internal medicine interns and residents in regular rotation at the hospital. In this capacity I was responsible for planning internal medicine grand rounds, establishing a core curriculum lectureship for the internal medicine residents and attending a daily morning report. I made regular teaching rounds with the house staff, several months of the year. I sat on the internal

medicine resident selection committee at UTMSH. I did this through the tenure of two Chairmen of medicine at UTMSH, the late Dr. Walter Kyrkendall and the late Dr. Thomas Andreoli. After about 12 or 15 years, UTMSH gradually withdrew its residents from St. Joseph Hospital. I remained in charge of the St. Joseph Hospital Transitional Internship and the teaching program for these individuals. As the Academic Chief of Medicine in the hospital, I continued to be responsible for educational activities, until my move to TMH about 12 years ago. Prior to this move, I assumed the position of Director of Medical Education at St. Joseph Hospital, and for our 5 residency programs with a total of about 65 house officers. I continued to be involved in recruitment lecturing, and served on a variety of Medical Education committees. For a time I was in charge of Continuing Medical Education (CME) at St. Joseph Hospital. The St. Joseph Hospital training programs were transferred to HMH in 2006, and at HMH I continued as the Director of the Transitional Residency, Program, which was closed in 2016, and I continue as one of four key faculty members for the Internal Medicine Residency Program and recently appointed for a brief term as Director of the Internal Medicine Residency Program pending a full time Director which has now been accomplished. I am the chairperson of the HMH IRB # 2 which must approve, review and monitor review research on human subjects.

From time to time I round as the attending physician of record on the medical wards with the house staff teams assigned to our University Teaching Service. In this capacity I have had the opportunity to teach the Weill Cornell, UTMB, Texas A & M and other medical students who have taken rotations at HMH and are assigned to these teams. At times, I also supervise the HMH Internal Medicine Resident's Continuity Clinic.

Clinical Care	***Dates***
Clinical Practice, Houston Methodist Hospital	May 2006 - Present
Private Practice, Surgical Oncology Consultants of Houston, LLP	June 2005 – May 2006
Private Practice, Stehlin & DeIpolyi Oncology Clinic	1985-2005
Private Practice, Christus St. Joseph Hospital Medical Hematology	July 1975 – July 1985
Private Practice, The Methodist Hospital Medical Hematology	July 1970 – June 1975

Administrative duties (including committees)	***Dates***
Transitional Year Residency Milestone Review Committee of the Accreditation Council for Graduate Medical Education (ACGME)	Jan.2011-2013
Chairman, IRB # 2, HMH	2009- Present
Chairman, Internal Medicine Review Committee Christus St. Joseph Hospital Houston, Texas	1980 - 2006
Chairman, Blood Utilization Committee Christus St. Joseph Hospital Houston, Texas	1976 - 2006

Chairman, Credentials Committee Houston Metropolitan PHO	2002- 2005
Christus St Joseph Hospital Medical Research Committee (IRB)	
Chairman	1988 -1990
Vice Chairman	2002 – 2006

J. RESEARCH SUPPORT

Additional IRB approved research, where I am a co-investigator at TMH includes protocols involving therapeutic bone marrow stem harvests for use in stimulation of arterial growth. My percent research effort on these studies is < 2 %. I am not directly funded by these studies.

PATENTS

Stehlin, JS, **Natelson, EA**, Giovanella, BC, Kozielski, AJ. Method for treating pancreatic cancer in human with water insoluble S-camptothecin of the closed lactone ring and derivates thereof. United States Patient No. 6,080,751 issued June 27, 2000

K. EXTRAMURAL PROFESSIONAL RESPONSIBILITIES

Trustee Stehlin Foundation for Cancer Research Houston Texas	1985- 2014
Trustee Gulf Coast Regional Blood Center Houston, Texas	2002- Present
California Rare Fruit Growers	1985 - Present
North American Fruit Explorers (NAFEX)	1985 – Present
President	1995-2014
Southern Fruit Fellowship (SFF)	1990 – Present
President	1998 - Present
Urban Harvest (Member, Advisory Board)	1997 - Present

BraeBurn Acres Civic Club (President 1983-87, 1989-1991, 2003-2005, 2012-2018)

Editorial Positions

Editor, Gulf Coast Fruit Study Newsletter	2001 – 2016
Editor, Southern Fruit Fellowship Newsletter	2012 - 2014

Reviewer

Leukemia	2009
Annals of Internal Medicine	2005
Archives of Internal Medicine	2005

Critical Reviews in Toxicology	2009
Haematologica	2010
Anti-Cancer Drugs	2010
International Blood Research & Reviews	2013
Critical Reviews in Toxicology	2014
Dove Press	2015
Int. J. Medicine and Health Development	2016

L. BIBLIOGRAPHY

Peer Reviewed Articles

1. **Natelson EA,** Lynch EC, Hettig RA, Alfrey CP Jr. Histiocytic medullary reticulosis. The role of phagocytosis in pancytopenia. Arch Intern Med 122:223-29, 1968.

2. **Natelson EA**, Allen TA, Riggs S, Fred HL. Bloody ascites: diagnostic implications. Am J Gastroenterol 52:523-7, 1968.

3. Fred HL, **Natelson EA**. Selection of patients for pulmonary embolectomy. Dis Chest 56:139-42, 1969.

4. **Natelson EA**, Lynch EC, Alfrey CP Jr, Gross JF. Heparin induced thrombocytopenia: an unexpected response to treatment of consumption coagulopathy. Ann Intern Med 71:1121-5, 1969.

5. **Natelson EA,** Duncan WC, Macossay CR, Fred HL. Amyloidosis palpebrarum. Arch Intern Med 125:304-07, 1970.

6. Dickson J, **Natelson EA,** Fred HL. Myxedema ascites. Am Fam Physician GP 1:93, 1970.

7. **Natelson EA,** Watts HD, Fred HL. Cystic medionecrosis of the pulmonary arteries. Chest 57:333-35, 1970.

8. Friedwald VE, Davis P, Roehm J Jr, Fechner RE, **Natelson EA**, Lane M, Spjut JH. Clinicopathological conference. Tex Med 66:78-85, 1970.

9. **Natelson EA**, Lynch EC, Hettig RA, Alfrey CP Jr. Acquired Factor IX deficiency in the nephrotic syndrome. Ann Intern Med 73:373-78, 1970.

10. **Natelson EA**, DeLallo LJ, Coltman CA Jr. Isolation of human factor IX by immunoadsorption. J Lab Clin Med 78:846, 1971.

11. Honig GR, **Natelson EA**. Factor IX deficiency in nephrosis. Ann Intern Med 74:298-9, 1971.

12. Cohen AA, **Natelson EA,** Fechner RE. Fibrosing interstitial pneumonitis in ankylosing spondylitis. Chest 59:369-71, 1971.

13. **Natelson EA**, Blumenthal BJ, Fred HL. Acute mercury vapor poisoning in the home. Chest 59:677-8, 1971.

14. **Natelson EA**, Lynch, EC, Britton, HA, Alfrey, CP Jr. Polycythemia vera in childhood. A case with chromosomal abnormality, immunoglobulin deficiency and chronic consumption coagulopathy. Am J Dis Child 122:241-4, 1971.

15. Girard DE, Carlson V, **Natelson EA,** Fred HL. Pneumomediastinum in diabetic acidosis: comments on mechanism, incidence and management. Chest 60:455-9, 1971.

16. Fred, HL, **Natelson EA**. Gonococcal dermatitis. Human Sexuality 6:183-191, 1972.

17. Cyprus GS, **Natelson EA.** Hematological manifestations of systemic lupus erythematosus. St Jos Hosp Med Surg Bull 8:193-202, 1973.

18. **Natelson EA,** Dooley DF. Rapid determination of fibrinogen by thrombokinetics. Am J Clin Pathol 61:828-33, 1974.

19. Brown CH III, **Natelson EA,** Bradshaw MW, Williams TW Jr, Alfrey CP Jr. The hemostatic defect induced by carbenicillin. New Engl J Med 265-70, 1974.

20. Kidd CR, Fred HL, **Natelson EA**. Bilateral hilar adenopathy in amyloidosis. New Engl J Med 290:972, 1974.

21. Glover CJ, McIntire LV, Leverett LB, Hellums JD, Brown CH III, **Natelson EA**. Effect of shear stress on clot structure formation. Trans Am Soc Artif Intern Organs 20B:463-8, 1974.

22. Brown CH III, Weisberg RJ, **Natelson EA,** Alfrey CP Jr. Glanzmann's thrombasthenia: assessment of the response to platelet transfusions. Transfusion 15:124-31, 1975.

23. Rahman F, Zanger B, **Natelson EA**. Factor IX deficiency in the nephrotic syndrome: studies with prothrombin complex concentrate. J Urol 113:853-5, 1975.

24. **Natelson EA,** Shapiro DM, Smith DF. Detection of mild Factor VIII deficiency by thrombocytokinetics. Am J Clin Path 64:95-100, 1975.

25. Brown CH III, **Natelson EA**, Bradshaw MW, Alfrey CP Jr, Williams TW Jr. A study of the effects of ticarcillin on blood coagulation and platelet function. Antimicrob Agents Chemother 7:652-7, 1975.

26. Glover CJ, McIntire LV, Brown CH III, **Natelson EA**. Dynamic coagulation studies: influence of normal and abnormal platelets on clot structure formation. Thromb Res 7:185-98, 1975.

27. Glover CJ, McIntire LV, Brown CH III, **Natelson EA**. Dynamic coagulation studies. Effects of fibrinogen concentration, Factor XIII deficiency and Factor XIII inhibition. J Lab Clin Med 85:644-56, 1975.

28. **Natelson EA**, Cyprus GS, Hettig RA. Absent Factor II in systemic lupus erythematosus. Immunological studies and response to corticosteroid therapy. Arthritis Rheum 19:79-82, 1976.

29. Shapiro DM, Zeluff GW, Wilson L, **Natelson** EA, Lynch EC. Thrombotic thrombocytopenic purpura despite absence of the spleen. report of a case. Am J Osteopath 75:798-802, 1976.

30. Siebert WT, **Natelson EA**. Chronic consumption coagulopathy accompanying abdominal aortic aneurysm. Arch Surg 111:539-41, 1976.

31. **Natelson EA**, Brown CH III, Bradshaw MW, Alfrey CP Jr, Williams TW. Influence of cephalosporin antibiotics on blood coagulation and platelet function. Antimicrob Agents Chemother 9:91-3, 1976.

32. Noon GP, Solis RT, **Natelson EA**. A simple method of intraoperative autotransfusion. Surg Gynecol Obstet 143:65-70, 1976.

33. Brown CH III, Bradshaw MW, **Natelson EA**, Alfrey CP Jr, Williams TW. Defective platelet function following the administration of penicillin compounds. Blood 47:949-56, 1976.

34. **Natelson EA**, Fred HL. Lead poisoning from cocktail glasses. Observations on two patients. JAMA 236:2527, 1976.

35. **Natelson EA**. Sustained thrombocytosis: clinical significance and therapeutic considerations. St Joseph Hosp Med J 11:109-17, 1976.

36. Moake JL, Cimo PL, Peterson DM, Roper P, **Natelson EA**. Inhibition of ristocetin-induced platelet aggregation and agglutination by vancomycin. Blood 50:397-406, 1977.

37. Fred, HL, Blakely, RW, **Natelson EA**. Progressive swelling of abdomen and lower extremities with abnormal chest roentgenogram. Chest 71:381-2, 1977.

38. Glover CJ, McIntire LV, Brown CH III, **Natelson EA**. Mechanical trauma effect on clot structure formation. Thromb Res Suppl 10:11-25, 1977.

39. Fred HL, **Natelson EA**. Grossly bloody urine of runners. South Med J 70:1394-96, 1977.

40. Cimo PL, Moake JL, Gonzales MF, **Natelson EA**, Fox KR. Inherited combined deficiency of Factor V and Factor VIII: report of a case with normal Factor VIII antigen and ristocetin-induced platelet aggregation. Am J Hematol 2:385-91, 1977

41. Zeluff GW, **Natelson EA**, Jackson D. Thrombocytopenic purpura - idiopathic and thrombotic. Heart & Lung 7:327-33, 1978.

42. **Natelson EA.** Treatment of iron overload in patients with chronic anemias. St Joseph Hosp Med J 13:78-83, 1978.

43. Crook JE, Woosley RL, Leftwich RB, **Natelson EA**. Agranulocytosis during combined antiarrhythmic therapy with procainamide and phenytoin. South Med J 72:1599-601, 1979.

44. **Natelson EA.** Current concepts of anticoagulant therapy. St Joseph Hosp Med J 14:77-86, 1979.

45. Novy SR, **Natelson EA**, Whitlock SL. Gaucher's disease in black adults. Am J Roentgen 133:947-49, 1979.

46. Kuntamukkula MS, McIntire LV, **Natelson EA**. A rheological study of the kinetics of coagulation in normal and hemophilic blood plasma. Biorheology 16:403-10, 1979.

47. **Natelson EA**, Siebert WT, Williams TW, Bradshaw MW. Combined effects of ticarcillin and cefazolin on blood coagulation and platelet function. Am J Med Sci 278:217-21, 1979.

48. Gentry LO, Jemsek JG, **Natelson EA**. The effects of sodium piperacillin on platelet function in normal volunteers. Antimicrob Agents Chemother 19:532-3, 1981.

49. **Natelson EA**. An update on blood and component therapy. St Joseph Hosp Med J 16:41-46, 1981.

50. **Natelson EA.** Hemorrhagic and thrombotic disorders. St Joseph Hosp Med *J* 18:5-18, 1983.

51. **Natelson EA.** Human blood coagulation: clinical and laboratory correlation. Clin Physiol Biochem 1:214-24, 1983.

52. Seider M, **Natelson EA,** Sher M. Extrahepatic biliary obstruction in multiple myeloma. Houston Med J 1:31-34, 1985.

53. **Natelson EA**, White D. Recurrent thrombotic thrombocytopenic purpura (TTP) in early pregnancy: effect of uterine evacuation. Obstet Gynecol 66:(3 Suppl) 54S-56S, 1985.

54. Sudarshan A, **Natelson EA,** Gordon C. Hereditary hemorrhagic telangiectasia and Factor VIII inhibitor. South Med J 78:623-4, 1985.

55. Gentry, LO, Wood BA, **Natelson EA**. The effects of apalcillin on platelet function in normal volunteers. Antimicrob Agents Chemother 27:683-4, 1985.

56. **Natelson EA.** Current therapy of aplastic anemia. Houston Med J 1:53-59, 1985.

57. Stern LD, **Natelson EA,** Lee CK. Cardiac tamponade as the first clinical feature of multiple myeloma. Houston Med J 2:101-04, 1985.

58. Casso D, **Natelson EA**, Williams BV, Braden D. Splenosis: a case report. Houston Med J 2:141-44, 1986.

59. **Natelson EA**, Bullock AC Jr, Siebert WT, Kupor LR: Recovery from allopurinol-induced agranulocytosis. Hou Med J 3:93-96, 1987.

60. Farmer K, **Natelson EA,** Stephanou N. HELLP syndrome as the only manifestation of preeclampsia: therapeutic considerations. Hou Med J 4:37-40, 1988.

61. Stehlin JS, Greeff PJ, de Ipolyi PD, Giovanella BC, Klein G, McGaff CJ, Davis BR, Williams LJ, **Natelson EA,** Anderson RF. Heat as an adjuvant in the treatment of advanced melanoma: an immune stimulant? Hou Med J 4:53-61, June 1988.

62. Natelson S, **Natelson EA**. Preparation of D-, DL- and L-homoserine lactone from methionine. Microchem *J* 40:116-32, 1989.

63. **Natelson EA**, Young DJ, Greeff PJ. Splenectomy in paroxysmal nocturnal hemoglobinuria. Houston Medicine 6:186-88, 1990.

64. Pantazis P, Mendoza JT, Early JA, Kozielski AJ, **Natelson EA,** Giovanella BC. 9-Nitro camptothecin delays growth of U-937 leukemia tumors in nude mice and is cytotoxic or cytostatic for human myelomonocytic leukemia cell lines *in vitro*. Eur J Haematol 50:81-9, 1993.

65. Hinz HR, Harris NJ, **Natelson EA,** Giovanella BC. Pharmacokinetics of the *in vivo* and in vitro conversion of 9-nitro-20 (S)-camptothecin to 9-amino-20 (S)-camptothecin in human, dog and mouse. Cancer Res 54:3096-100, 1994.

66. Natelson S, Pantazis P, **Natelson EA**. L-homoserine hydroxamic acid as an anti-tumor agent. ***Clin Chem Acta*** 229:133-45, 1994.

67. **Natelson EA,** Fred HL. Fever, leukocytosis and an enlarging flank mass. Hosp Pract 29(4):33-4, 1994.

68. Tschen LF, Tschen JA, **Natelson EA**. Photophobia and thickened eyelids in a healthy-appearing nurse. Hosp Pract 31(2):73-4, 1996.

69. Giovanella BC, **Natelson EA**, Harris N, Vardeman D, Stehlin JS. Protocols for the treatment of human tumor xenografts with camptothecins. Ann N Y Acad Sci 803:181-87, 1996.

70. **Natelson EA**, Giovanella BC, Verschraegen CF, Fehir KM, de Ipolyi PD, Harris N, Stehlin JS. Phase I clinical and pharmacological studies of 20-(S)-camptothecin and 20-(S)-9-nitrocamptothecin as anticancer agents. Ann N Y Acad Sci 803:224-30, 1996.

71. Verschraegen CF, **Natelson EA**, Giovanella BC, Kavanagh JJ, Kudelka AP, Freedman RS, Edwards CL, Ende K, Stehlin JS. A phase I clinical and pharmacological study of oral 9-nitrocamptothecin, a novel water-insoluble topoisomerase-I inhibitor. Anticancer Drugs 9:36-44, Jan 1998.

72. Trizna Z, Tschen J, **Natelson EA**. Multiple subcutaneous nodules on the torso and leg. Arch Dermatol 134:1479,1482, 1998.

73. Gupta E, Toppmeyer D, Zamek R, Much J, Ibrahim N, Goodin S, Kane M, Aisner J, Li X, Haluska Jr P, Medina M, Cornfield A, **Natelson EA,** Giovanella BC, Saleem A, Rubin E. Clinical evaluation of a strategy of sequential topoisomerase poisoning in the treatment of advanced malignancy. Cancer Therapeutics 1:292-301, 1998.

74. Stehlin JS, Giovanella BC, **Natelson EA**, de Ipolyi PD, Coil D, Davis B, Wolk D, Wallace PJ, Trojacek A. A phase II study of 9-nitrocamptothecin (RFS-2000) in patients with advanced pancreatic cancer: A preliminary report. Internat J Oncol 14:821-31, 1999.

75. Trizna Z, Tschen J, **Natelson EA**. Atypical mycobacterial infection in a patient with hairy cell leukemia. Cutis 67:241-2, 2001.

76. **Natelson EA**. Myelodysplasia with isolated trisomy 15: A 15-year follow-up without specific therapy. Am J Med Sci 331:157-158, 2006.

77. **Natelson EA.** Pregnancy-induced pancytopenia: Distinctive hematological features. Am J Med Sci 332:205-207, 2006.

78. **Natelson, EA.** Benzene-induced acute myeloid leukemia – a clinician's perspective. Am J Hematol 82:826-830, 2007.

79. **Natelson, EA.** Benzene and Sideroblastic Erythropoiesis – Is There an Association? Am J Med Sci 334:356-360, 2007.

80. Pyatt, D, Golden R., **Natelson EA.** Is inhalation exposure to formaldehyde a biologically plausible cause of lymphohematopoietic malignancies. Regulatory Toxicology and Pharmacology 51:119-133, 2008.

81. **Natelson EA**, Pyatt D. Temozolomide-Induced Myelodysplasia. Advances in Hematology (an on-line journal) Volume 2010, Article ID 760402,doi:10.1155/2010/760402.

82. Muhyieddeen K, **Natelson EA**. Atypical Ringed Sideroblasts in Association with Trisomy 19 and Myelodysplasia. Comp Clin Pathol 2012;21:223-225, DOI: 10.1007/s00580-011-1298-0

83. **Natelson EA.** Extreme thrombocytosis and cardiovascular surgery: Risks and management. Texas Heart Institute Journal 2012;39:(6) 792-798.

84. Ahn I, **Natelson EA**, Rice L. Successful long-term treatment of Philadelphia chromosome negative myeloproliferative disorders with combination of hydroxyurea and anagrelide. Clin Lymp Myeloma Leuk 2013;13 (S2):S300-304.

85. **Natelson EA**, Pyatt, D. Acquired myelodysplasia (AMD) or myelodysplastic syndrome (MDS) - Clearing the fog. Advances in Hematology 2013; e-published, http//dx.doi.org/10.1155/2013/309637.

86. **Natelson EA**. Skepticism, the Trained Eye, and the Telltale Clue. Texas Heart Institute Journal. 2016;43:205-206.

87. Mundt, KA, Gallagher, AE, Dell LD, **Natelson EA**, Boffetta, P, Gentry PR. Does occupational exposure to formaldehyde cause hematotoxicity and leukemia-specific chromosome changes in cultured myeloid progenitor cells? Critical Reviews in Toxicology 2017;47(7):592-602.

88. Mundt KA, Gallagher AE, Dell LD, **Natelson EA**, Boffetta P, and Gentry PR. Formaldehyde, hematotoxicity and chromosomal changes. Can Epidemiol, Biomark & Prev, Letter to the Editor. 2018;27:119.

89. Mundt KA, Gallagher AE, Dell LD, **Natelson EA**, Boffetta P, Gentry PR. Response to Bernard D. Goldstein's Letter to the Editor. Crit Rev Toxicol doi.org/10.1080/10408444.2018.1431765.

Book chapters

1. Fred HL and **Natelson EA.** Pulmonary thromboembolism. In: Crises in Medicine, Chapter 5, pp 56-75. Eichorn RD and Beard EF (eds). *Chas C. Thomas: Springfield*, Springfield, Illinois, 1973.

2. Natelson S and **Natelson EA.** Principles of applied clinical chemistry: chemical background and medical applications, Vol. I., Maintenance of Fluid and Electrolyte Balance. Plenum Press, New York, 1975. 393 pp.

3. *Ibid.* Vol. II. The erythrocyte: chemical composition and metabolism. Plenum Press, 1978. 560 pp.

4. *Ibid.* Vol. III. Plasma proteins in nutrition and transport. Plenum Press, 1980. 554 pp.

5. Natelson S and **Natelson EA.** Regulation of water and electrolyte balance. In: CRC Handbook of Clinical Chemistry, Vol. IV, p. 3-42. Werner M., (ed). CRC Press Inc., Boca Raton, Florida, 1989.

6. Stehlin JS, **Natelson EA**, Hinz HR, Giovanella BC, de Ipolyi PD, Fehir KM, Trezona TP, Vardeman DM, Harris NJ, Marcee AK, Kozielski AJ and Ruiz-Razura A. Chapter 5 in: Camptothecins: New Anticancer Agents, pp. 59-65. Potmesil M and Pinedo H (eds). CRC Press Inc., Boca Raton, Florida, 1995.

7. Giovanella BC, **Natelson EA**, Harris N, Vardeman D and Stehlin JS. Protocols of the treatment of human xenografts with camptothecins. In: The Camptothecins From Discovery to the Patient. Pantazis P, Giovanella BC, Rothenberg ML, (eds). Ann NY Acad. Sci, 803:181-87, New York, 1996.

8. **Natelson EA**, Chugtai-Harvey, I and Rabbi, S. Overview of Clinical Hematology, Chapter 39 in Textbook of Family Medicine, 9th Edition, Rakal, R., Ed., Elsevier Press, 2016, published March, 2015.

TEACHING VIDEOCASSETTES

1. **Natelson EA** and Williams TW. Critical decisions in medicine. #7 Park Row Publishers, New York, NY, 1984.

ABSTRACTS

Ahn IE, **Natelson, EA**, Rice L. Combination treatment of essential thrombocythemia with hydroxyurea and anagrelide. Blood 2011;118:Abstract 5177.

SELECTED HORTICULTURAL ARTICLES

1. **Natelson, EA**. More on fireblight control. Pomona 22(4):36-7, 1989.
2. **Natelson, EA.** Low chill pears. Southern Fruit Fellowship Newsletter. 33:8-9, 1996.
3. **Natelson, EA**. Pear and apples together. Pomona 31(1):56, 1998.
4. **Natelson, EA**. Pear interstem length. Pomona 31(3):25-27, 1998.
5. **Natelson, EA.** Pear interstems – how long is long enough. Southern Fruit Fellowship Newsletter 40:6, 1998.
6. **Natelson, EA**. In search of Jean-Baptiste Van Mons 1765-1842. Pomona 34:50-54, 2001.
7. **Natelson, EA.** Dwarfing pear trees in the South. Pomona 35:19-20, 2002.
8. **Natelson, EA.** New Plums for Old. Southern Fruit Fellowship Newsletter 60:8-9, 2003.

9. **Natelson, EA.** Lessons from the past. Southern Fruit Fellowship Newsletter 58:6-9, 2002.

10. **Natelson, EA.** Sweet cherries for the South. Southern Fruit Fellowship Newsletter. March 2007.

11. **Natelson, EA.** The fragrant pear. Southern Fruit Fellowship Newsletter April-June, Issue 76, 2007.

12. **Natelson, EA.** Observations on pears grafted to quince in zone 9. Southern Fruit Fellowship Newsletter 84:12-13, 2009.

13. **Natelson, EA**. The long wait for the Bloomsweet grapefruit. Pomona 44:66-68, 2011.

14. **Natelson, EA.** Air Layering – A new wrinkle. Southern Fruit Fellowship Newsletter, Issue 95:3-4, Jan-Mar 2012.

15. **Natelson, EA.** Dwarfing citrus rootstock. Pomona 46:61-63, 2013.

17. **Natelson, EA.** What Old Home never told Farmingdale. Pomona 47:18-19, 2014.

Signature:_____

Date:_____

Prior Five Years Testimony List of Dr. Ethan A. Natelson

The following is a list of all cases in which I have testified at trial or by deposition, since April 30-2014.

1. *Dean Charles Tessitore and Tammy Tessitore vs.* Chevron Oronite Company, LLC, et al.; Civil Action No. 06-4041; In the Civil District Court for the Parish of Orleans, State of Louisiana. Case had to do with allegations of causation in an individual with Primary Mediastinal Non-Hodgkin lymphoma. Testified by deposition for the defense on 04-30-2014 and at trial on 07-21-2014.

2. *Jim Willis and Cathy Willis v.* Shell Oil Company, et al., Cause No. 2012-52003; In the District Court of Harris County, Texas, 80th Judicial District. Case had to do with allegations of benzene exposure via exposure to fuels as a cause of acute lymphoblastic leukemia (ALL). Testified for the defense on 08-11-2014, by deposition and at trial at 10-22-2014.

3. *Yolanda Burst Individually, and as the Legal Representative of Bernard Ernest Burst, Jr.* v. Shell Oil Co., et al.; Cause No. 2014 cv 109A2; In the District Court for the Eastern District of Louisiana. Testified for the defense by deposition on 11-17-2014, and at a Daubert hearing before the Court on 02-11-2015. The case had to do with allegations of AML consequent to gasoline exposure. On appeal, the case was ruled in favor of the defense.

4. *Virgil Hood and Lorrie Hood, v.* E.I. DuPont de Nemours & Company, et al., Cause No. DC-03619. In the District court of Dallas County, Texas, 160th Judicial District. Testified for the defense by deposition on 02-23-2015 and at trial on 10-13-2015. Case had to do with allegations of AML caused by trace benzene present in paints, fuels and solvents. On appeal the case was ruled in favor of the defense.

5. *Cheri Dahlin, et al., vs. Archer-Daniels-Midland Company, et al.;* CA D:14-CV-00085-CMR-HCA, In the United States District Court, Southern District of Iowa, Davenport Division. Case had to do with allegation of causation of AML in a patient with long-standing HIV disease. Testified for the defense at trial on 04-01-2016. On appeal, the case was ruled in favor of the defense.

6. *Donald Johnson, et al., versus Motiva Enterprises, LLC, Equilon Enterprises, LLC Riverbend Shell, Inc. And Diane Williams:* No. 2003-5123 Sect 6 "L", c/w 2003-6746, in the Civil District Court for the Parish of Orleans, State of Louisiana. Testified for the defense by deposition on 01-13-2017 and at a Bench trial on 02-16-2017. Case had to do with allegations of various illnesses including anemia caused by alleged inhalation of gasoline vapors from a leaking service station storage tank, into a sewer drainage line which communicated with a restaurant.

7. *Michael Butts and April Butts, h/w vs. United States Steel Corporation, et al.*, Court of Common Pleas, January Term, 2015, NO: 3561, Philadelphia County. Testified at trial for the defense on 05-15-2017. Case had to do with allegations of causation of AML by alleged occupational benzene exposure.

8. *Paul Simmons and Dianna Simmons v. Sunoco, Inc. (R&M), et al.* Court of Common Pleas, Philadelphia County, PA; No. 04219. Testified for the defense on 05-01-2018, by video-conference directly into the Courtroom. Case had to do with allegations of death by aplastic anemia consequent to benzene toxicity in a patient with heart failure from cardiac amyloidosis.

9. *Robert J. O'Donnell and Sandra O'Donnell v. W.F. Taylor Co., Inc., et al.* Case No.: 2013 CA 017987, Division AO, The Circuit Court for the Fifteenth Judicial Circuit in and for Palm Beach County, Florida. Testified for the defense on August 8, 2018. Case has to do with allegations of causation of RARS by remote trace benzene exposure as a carpet layer.

10. *Charles Nelson, III and Jean Nelson v. BP Products North America Inc., et al., USDS for the Eastern District of Pennsylvania, Docket No.:2:16-cv-4888.* Testified for the defense on October 10, 2018. Case has to do with allegations of causation of atypical CML (aCML) by alleged exposures to benzene.

11. *Randy Eaves and Jessica Eaves, et al., versus Ashland, Inc., Union Oil Company of California, Shell Oil Company, Inc, et al.*, Case No.*:MSC17-00850 (Related to Case No. C16-00815), In the Superior Court of the State of California for the County of Contra Costa.* Testified for the defense on November 12, 2018 and at trial on March 29, 2019. Case has to do with causation of a case of JAK-2 positive primary myelofibrosis by alleged remote exposures to benzene

12. *Darryl W. Deaton (Executor for the Estate of Walter C. Deaton) and Kristi D. Patterson (Executrix for the Estate of Walter C. Deaton) v. United States Steel Corporation, et al.;* In the Court of Common Pleas, County of Charleston, South Carolina. Testified for the defense on 05-17-2019 by deposition. Case has to do with allegations of AML caused by alleged benzene exposures.

EXHIBIT C

EPI

8805 Columbia 100 Pkwy
Suite 100
Columbia, MD 21045

(410) 740-9600
FAX (410)740-9606

www.episervices.com

22 July 2019

EXPERT REPORT OF JOHN W. SPENCER, CIH, CSP

Re: Mary Webb, individually, and as the representative of The Estate of Elwyn Webb v. SFPP, et al.
EPI Project No. 219272

This summary report has been prepared at the request of the defendant companies listed below,[1] as a synopsis of my opinions regarding Mr. Elwyn Webb's (deceased) potential benzene exposures while working as a tank truck driver for Calzona Tankways (Calzona) and later for Coastal Transportation Company, Inc. (Coastal) during the loading of fuel products between 1985 and 2015. According to the Complaint, Mr. Webb was diagnosed with Myelodysplastic Syndrome (MDS) in January 2016 and died on 19 March 2016. [1]

Mr. Webb smoked approximately 1 pack per day of Camel Lights from 1978 to 2006 or 2008. [2] [3]

I have been an industrial hygienist for 42 years. Currently, I am President of Environmental Profiles, Inc. in Baltimore, Maryland. Formerly, I was with the National Institute for Occupational Safety and Health and led a group of industrial hygienists conducting research for the National Occupational Exposure Survey. As an industrial hygienist for the United States Coast Guard, I conducted numerous exposure assessments of a wide range of benzene-containing products. My responsibilities also included the management of the occupational medical monitoring program for the 5th Coast Guard District. I was President of the Chesapeake Section of the American Industrial Hygiene Association (AIHA) and was a member of the national AIHA Product Health and Safety Committee and the Emergency Response Planning Committee. I have also authored the *Health and Safety Audits Manual*, published by Government Institutes, and the *AIHA Hazard Communication Guide*, published by the AIHA. I have published several articles in peer-reviewed journals on airborne exposure modeling and assessment. In these papers, I have discussed the value and limitations of exposure assessment models.

The American Board of Industrial Hygiene certifies me as an industrial hygienist and the Board of Certified Safety Professionals certifies me as a safety professional.

A copy of my curriculum vitae is included as Attachment 1. Environmental Profiles, Inc. charges $295 per hour plus expenses for my time in preparation and testimony in this matter.

---

[1] ConocoPhillips Company, Kinder Morgan/SFPP, Caljet of America, Circle K Terminal, Pro-Petroleum, Inc., Valero Marketing and Supply Company

Employee Work History

Mr. Webb is deceased. Consequently, the only information regarding Mr. Webb's work history was obtained from the deposition of his wife, Mary Major, [3] his son, Joshua Webb, [4] his coworkers, Eugene Martin, [5] Jimmy Melvin, [6] and Robert Super, [7] the Vice President of Safety for Coastal Transport Company, Barry Detlefsen, [8] and Mr. Webb's employment records from Calzona/Coastal. [9]

Mr. Webb was born on 23 December 1950 and graduated from high school in May 1969. [10] On his Calzona Application for Employment dated 24 January 1985, Mr. Webb listed former employers to include Hiway Trailer Services (January 1974 to October 1982), Transport West (October 1982 to December 1984), and Whitfield Tank Lines (7 January 1985 to 23 January 1985). [10] He noted that he had experience hauling bulk hazardous materials for eight years, from January 1974 to October 1982. [10]

According to Ms. Major, she met Mr. Webb sometime in 1981 when they both worked for Whitfield Tank Lines (Deposition of Mary Major, page 8). Mr. Webb had been working for Whitfield Tank Lines when she began working there in 1981 and she did not know how long he had been employed (Deposition of Mary Major, page 8). Mr. Webb was a truck driver for Whitfield Tank lines and he hauled fluids including gasoline, sulfuric acid, and other chemicals (Deposition of Mary Major, page 9). No further details regarding the Mr. Webb's employment at Whitfield Tank Lines were available including information about where he picked up and unloaded products and the frequency and duration of those tasks. Whitfield Tank Lines is no longer in business (Deposition of Mary Major, page 9).

After working for Whitfield Tank Lines, Mr. Webb hauled dry goods for Transportation West (Deposition of Mary Major, page 10). Ms. Major did not know how long he worked for Transportation West (Deposition of Mary Major, page 10).

Other jobs Mr. Webb performed included installing carpet, working as a mechanic for three months at Transportation West, and driving a forklift (Deposition of Mary Major, pages 16-17). No additional details were provided.

Mr. Webb began his employment as a tank truck driver for Calzona on 1 March 1985 (Deposition of Barry Detlefsen, page 18). Coastal acquired Calzona on or about 1 January 1994, at which time Mr. Webb began employment at Coastal (Deposition of Barry Detlefsen, page 18). [11] Mr. Webb continued his employment as a tank truck driver with Coastal through 2 July 2015. [11]

Mr. Robert Super was a coworker of Mr. Webb and worked as a tank truck driver for Calzona from 1979 to 1993 and then continued with Coastal following the Calzona acquisition until 2002 (Deposition of Robert Super, pages 15-16). According to Mr. Super, he and Mr. Webb hauled the same products which included chlorine, hydrochloric acid, liquid fertilizer, alcohol, and

petroleum products including diesel fuel, different grades of gasoline, jet fuel, and avgas (Deposition of Robert Super, pages 16-17; 26; 32). According to Mr. Super, there were a lot of drivers that only wanted to deliver gasoline or diesel (Deposition of Robert Super, page 37). He and Mr. Webb were two drivers that were willing to deliver fuel products and chemicals (Deposition of Robert Super, page 37).

Mr. Super and Mr. Webb generally worked the same shift and Mr. Super saw Mr. Webb at different loading racks or at different customer locations (Deposition of Robert Super, pages 19-20). Mr. Super occasionally worked on a sleeper team with Mr. Webb and a large part of this work involved the delivery of lubricating oils (Deposition of Robert Super, page 21). According to Mr. Super, this was not steady work and he estimated that this occurred once or twice per month during employment with Calzona and Coastal (Deposition of Robert Super, pages 21-22).

Mr. Super testified that all drivers, including Mr. Webb wanted to deliver to areas outside the metropolitan Phoenix area because of the traffic (Deposition of Robert Super, page 23). He recalled that Mr. Webb did a lot of work down in Tucson and Flagstaff, Arizona and areas toward California and into Mexico (Deposition of Robert Super, pages 23-24). Mr. Super estimated that approximately 25% to 30% of Mr. Webb's trips were outside the metropolitan area (Deposition of Robert Super, page 25). During the last five years that they worked together (1998-2002), Mr. Webb was a dedicated driver for Love's Truck Stops, delivering diesel fuel and gasoline (Deposition of Robert Super, page 29). Most of those truck stops were outside the metropolitan Phoenix area (Deposition of Robert Super, page 91). When Mr. Webb worked for Calzona and Coastal, he transported more fluids outside the metro areas as compared to his coworkers (Deposition of Mary Major, page 26; Deposition of Jimmy Melvin, page 102).

Mr. Jimmy Melvin was a coworker of Mr. Webb and worked as a tank truck driver for Calzona from 1984 to 1995 and then continued with Coastal following the Calzona acquisition until 1998 (Deposition of Jimmy Melvin, page 9). According to Mr. Melvin, Calzona was one of the premier fuel carriers (Deposition of Jimmy Melvin, page 11). In addition to hauling fuel products, Coastal, including Mr. Webb, also hauled some chemicals including bleach, muriatic acid, sulfuric acid, and concentrated soap during a period of four or five years (Deposition of Jimmy Melvin, pages 20-22; 31; 95-96). During the time that he worked with Mr. Webb between 1984 and 1995, 98% of loads of concentrated soap were hauled by Mr. Webb from Tempe, Arizona to Banning, California (Deposition of Jimmy Melvin, page 32). Those runs did not happen every week and when they happened it may be once per week (Deposition of Jimmy Melvin, page 33).

Mr. Eugene Martin was a coworker of Mr. Webb and worked with him as tank truck driver at Coastal from 1997 to 2011 (Deposition of Eugene Martin, page 9). There were occasions that he and Mr. Webb loaded at the same facility (Deposition of Eugene Martin, page 31). He never recalled seeing Mr. Webb have any issues or accidents at any of the terminals while he was loading products (Deposition of Eugene Martin, page 33). According to Mr. Martin, Mr. Webb made more out of town deliveries than other drivers (Deposition of Eugene Martin, page 155).

Fuel Product Information

This matter involved the transport of gasoline, diesel fuel, and avgas, not benzene. Gasoline is a complex mixture of paraffinic, olefinic, and aromatic hydrocarbons ranging from C_3 to C_{11} compounds and may contain as many as 250 separate hydrocarbons. [12] Overall, the mean benzene content of gasoline is 1% or less. [13] [12] Toxicity testing has been conducted on gasoline and, according to IARC (1989), regarding unleaded gasoline exposures, there is inadequate evidence of the carcinogenicity in humans and limited evidence for the carcinogenicity in experimental animals. [14] Gasoline is not included on OSHA's list of their recognized human carcinogens. Additionally, agencies such as NTP, IARC, ACGIH, and ATSDR, who review chemicals for carcinogenicity, have reviewed the gasoline literature and concluded that it does not cause any benzene related cancer even though it contained limited amounts of benzene. [14] [15] [12] [16] Additionally, a review of the industrial hygiene literature for gasoline failed to document any relationship with MDS (ACGIH Documentation of TLVs). [12]

Aviation gasoline (avgas) are special grades of gasoline for use in aviation reciprocating engines and included all refinery products within the gasoline range that were to be marketed straight or in blends. [14] Many of the gasoline requirements of the automotive engine are shared by gasoline powered aviation engines. [14] The same types of blending materials as those used in automotive gasolines may be used in aviation gasolines, but higher percentages of some stocks (especially alkylates) and additional tetraethyl lead are used to meet the higher octane requirements. [14] Relevant industrial hygiene references cited no association between avgas exposures and MDS. [14] [15]

Diesel fuel is a complex mixture of hydrocarbons produced by the distillation of crude oil. [14] It consists of hydrocarbons having carbon numbers predominately in the range of C9-C20. [14] Diesel fuels may also contain minor amounts of constituents such as n-hexane (below 0.1%), benzene (below 0.02%), toluene, xylenes and ethyl benzene (0.25-0.5%). [14] According to IARC (1989) distillate (light) diesel fuels are not classifiable as their carcinogenicity to humans. [14] Relevant industrial hygiene references cited no association between diesel fuel exposures and MDS. [14] [15] [17]

Most commercial jet fuels have basically the same composition as kerosene, but they are made under more stringent specifications than those of kerosene. [14] The basic components of kerosene used for aviation is the straight run kerosene stream, which consists of hydrocarbons with carbon numbers predominantly in the range of C9-C16 (C4-C16 for wide cut fuels) and which boil in the range of 150-290°. [14] Kerosene in the C9-C16 range normally has a boiling range well above the boiling point of benzene, accordingly the benzene content is normally below 0.02%. [14] Relevant industrial hygiene references cited no association between jet fuel exposures and MDS. [14] [15] [18]

OSHA has not developed a Permissible Exposure Limit (PEL) for gasoline, diesel fuel, jet fuel, or avgas. However, the ACGIH has developed gasoline and kerosene/jet fuel TLVs. The TLV "*...refers to airborne concentration of chemical substances and represents conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse health effects*" (ACGIH 2019). [19] The first gasoline TLV was developed in 1982. Since that time, the gasoline TLV was 300 ppm as an 8-hour time weighted average (TWA) with a short term excursion limit (STEL) of 500 ppm. [12] Table 1 contains a summary of the ACGIH TLV for gasoline. This gasoline TLV was recommended to minimize the potential for ocular and upper respiratory tract irritation and the STEL was recommended to minimize the potential for ocular and upper respiratory tract irritation and acute depression of the central nervous system. [12]

Table 1: Gasoline Occupational Health Standards and Guidelines

Year	OSHA Gasoline PEL		ACGIH Gasoline TLV	
	8-Hour TWA	STEL/Ceiling	8-Hour TWA	STEL/Ceiling
1982-Present	none	none	300 ppm	500 ppm

The first kerosene/jet fuel TLV was developed in 2003. [18] Since that time, the kerosene/jet fuel TLV was 200 mg/m^3 as an 8-hour TWA. Table 2 contains a summary of the ACGIH TLV for kerosene/jet fuel. Exposures to aerosols or high vapor concentrations can cause respiratory irritation and central nervous system depression. [18]

Table 2: Kerosene/Jet Fuels Occupational Health Standards and Guidelines

Year	OSHA Gasoline PEL		ACGIH Gasoline TLV	
	8-Hour TWA	STEL/Ceiling	8-Hour TWA	STEL/Ceiling
2003-Present	none	none	200 mg/m^3	none

OSHA recognized that potential benzene exposures at fuel distribution terminals that had vapor recovery systems were substantially below the OSHA benzene action level of 0.5 ppm. [20] "*Thus OSHA concluded that the use of either type of vapor control system would result in average exposures virtually always below the action level and proposed to exempt from this action loading operations at both bulk plants and terminals which use the vapor control systems on this basis.*"

Bulk Fuel Transportation Employment

Upon arrival at the Calzona/Coastal terminal the dispatcher sent the drivers to a particular location for loading/unloading liquid products (Deposition of Robert Super, page 30). Drivers could load at a single or multiple terminals in a day (Deposition of Eugene Martin, page 33). The hoses and fitting used to unload fuel products were owned by the trucking company and the hoses at the fuel terminal were owned by the terminal (Deposition of Eugene Martin, pages 34;

122). As part of the procedure prior to leaving the trucking terminal, the drivers were required to inspect that equipment and gaskets (Deposition of Eugene Martin, page 35; Deposition of Robert Super, pages 82-83). The drivers carried extra parts on the truck in case there was a problem with that equipment (Deposition of Eugene Martin, page 35).

Barry Detlefsen has been the vice president of safety at Coastal since approximately 2010 (Deposition of Barry Detlefsen, page 12). Coastal's business model was that the drivers made local deliveries so that they could be home at night (Deposition of Barry Detlefsen, page 24). On a system wide average, drivers hauled five to eight loads per day (Deposition of Barry Detlefsen, page 25). Drivers would spend less than 45 minutes at a rack, providing they were not waiting in line or if there was traffic congestion (Deposition of Barry Detlefsen, page 25).

Mr. Millican was a tank truck driver for Woodland Oil between 1983 and 1985 and a tank truck driver for Arco between 1985 and 1995 (Deposition of John Millican, pages 14-15). During that time, he loaded fuel at the Phoenix tank farm (Deposition of John Millican, page 16). Between 1983 and 1985 he was familiar with Calzona Tankways and noted that they had the largest number of petroleum tank trucks at that time (Deposition of John Millican, page 18). When he worked for Woodland and spent 75% of his time delivering locally and 25% of his time delivering outside Phoenix, he estimated that he loaded five to six loads per day (Deposition of John Millican, page 20). Mr. Millican estimated that it took approximately 15 minutes to load a truck (Deposition of John Millican, page 21). When he loaded at the Phoenix terminal in 1983 there was vapor recovery present but noted that Kinder Morgan/SFPP may have had one or two top loading racks for jet or diesel fuel (Deposition of John Millican, page 25). Mr. Millican had heard stories before that time that gasoline had been top loaded (Deposition of John Millican, page 26). He never bottom-loaded gasoline without vapor recovery at the Phoenix tank farm (Deposition of John Millican, page 26). Tank truck drivers performed similar tasks and the loading procedures were substantially similar from terminal to terminal (Deposition of John Millican, pages 30; 32).

According to Mr. Super, a coworker of Mr. Webb, the loading racks where Mr. Super and Mr. Webb loaded had vapory recovery systems between 1985 and 2002 (Deposition of Robert Super, page 110). He later stated that Mr. Webb would have top loaded at the various terminals between 1985 and 1990 (Deposition of Robert Super, pages 173-174). However, Mr. Super noted that when he started working for Calzona in 1979 he had to learn how to bottom load on a gasoline truck and noted that most of the Calzona fleet was equipped for bottom loading (Deposition of Robert Super, pages 232; 245). According to Mr. Super, he never saw Mr. Webb top loading gasoline at any facility (Deposition of Robert Super, page 258).

According to Mr. Melvin, a coworker of Mr. Webb, during his time while working for Calzona and Coastal between 1984 and 1998, there were no changes in the loading or unloading processes besides changes from the key system to the card system for access to the loading process (Deposition of Jimmy Melvin, pages 16-17). Mr. Melvin stated that between 1984 and 1985 the only loading rack that still was top loaded was the jet fuel rack and the rest were bottom

loading with vapor recovery (Deposition of Jimmy Melvin, page 38). Later, Mr. Melvin testified that all the gasoline racks had been converted to bottom loading racks by approximately 1981 or 1982 (Deposition of Jimmy Melvin, page 112).

Mr. Martin testified that by the time he began working in 1997, with the exception of the Caljet terminal, all terminals had vapor recovery for gasoline (Deposition of Eugene Martin, page 103). He stated that the Pro Petroleum rack did not have vapor recovery for its diesel loading, but they eventually (time not stated) added vapor recovery (Deposition of Eugene Martin, page 103).

Mr. Super testified that in 1985 the Environmental Protection Agency (EPA) required terminals to change from top loading racks to bottom loading racks and incorporate vapor recovery (Deposition of Robert Super, page 172). Furthermore, at the time that the EPA and Maricopa County regulations were incorporated they would not have performed top loading of gasoline, but did continue to top load for diesel and jet fuel (Deposition of Robert Super, page 234).

When arriving at a terminal and waiting at the loading rack ready line, the drivers were to turn off all lights and electrical items, check for flat tires, look at the loading rack and make sure the loading arms, scully cord, and vapor hoses were out of the way, and look to see if there was any liquid in the loading rack (Deposition of Robert Super, page 69; Deposition of Eugene Martin, pages 171-172). If there was liquid on the loading rack, or if leaks were observed in the equipment, the driver was supposed to report that finding to the terminal point person immediately (Deposition of Robert Super, page 71; Deposition of Eugene Martin, page 35; Deposition of Eugene Martin, page 173). Once the driver pulled onto the rack, they connected the ground line, and attached the vapor recovery and product hoses (Deposition of Robert Super, page 61). The drivers then entered the product they needed to load into the computer and stood by the meter during loading (Deposition of Robert Super, page 61; Deposition of Eugene Martin, page 25). They were required to stand there, so that in the event of an emergency they could press the stop button (Deposition of Robert Super, page 61; Deposition of Jimmy Melvin, page 57). If the vapor recovery was not working at a rack the terminal shut that rack down (Deposition of Robert Super, page 190; Deposition of Eugene Martin, page 105).

There were times when a truck would have to wait two to three hours to load (Deposition of Robert Super, page 131). The loading time varied from terminal to terminal. At the Shell terminal Mr. Super could pull into the loading rack, load, and get his bill of lading in about 20 minutes (Deposition of Robert Super, page 210). The average time it took to load at the terminal was approximately 30 minutes (Deposition of Robert Super, page 211).

Bills of lading were provided to the drivers by the terminal (Deposition of Robert Super, page 85). This document contained information on the products that were being transported (Deposition of Robert Super, page 86). When the driver completed his loading process, he walked into a small office space and printed the bill of lading (Deposition of Barry Detlefsen, page 156). The bill of lading was known as the shipper's hazardous material certification (Deposition of Barry Detlefsen, page 156). At this point, the driver had printed a document

which showed everything he needed to know about the handling and safety of this particular product that was onboard his cargo tank motor vehicle (Deposition of Barry Detlefsen, page 156). The truck did not leave the facility until the driver had the correct shipping papers (Deposition of Barry Detlefsen, page 156).

Between 1985 and 2002 there were no delivery locations in the metropolitan Phoenix area that did not have vapor recovery (Deposition of Robert Super, page 116; Deposition of Jimmy Melvin, page 85). Upon arrival at a delivery location, the driver sets up the perimeter cones and they get a printout from the store location and stick the fuel tanks in order to determine the amount of fuel in the tanks (Deposition of Robert Super, page 103). The bill of lading contained an entry area where the drivers were supposed to record the stick measurements before and after filling the tanks (Deposition of Robert Super, page 104). When performing this task, which took three or four minutes, the driver wore gloves (type not specified) and used a rag to clean the stick (Deposition of Robert Super, page 105).

During his career, Mr. Super noted that he had seen signs at loading racks and gasoline stations, which indicated that gasoline contained chemicals that were known to cause cancer (Deposition of Robert Super, page 122). Mr. Super also knew that gasoline and avgas contained benzene (Deposition of Robert Super, page 122). He also stated that it was common knowledge to him and hazmat drivers like Mr. Webb that a chemical like benzene was harmful to their health (Deposition of Robert Super, page 123). Mr. Super stated that it was early in his career that he learned that benzene was a component of gasoline (Deposition of Robert Super, page 210).

Mr. Super testified that during his daily activities when at the Calzona/Coastal terminal, when loading at terminals, when driving, and when unloading fuel product, he would not generally smell a gasoline odor (Deposition of Robert Super, page 101). When connecting or disconnecting fuel hoses he may detect a gasoline odor (Deposition of Robert Super, page 189; Deposition of Jimmy Melvin, page 62). Mr. Melvin and Mr. Martin testified that they could detect the odor of gasoline at every terminal where they worked (Deposition of Jimmy Melvin, page 59; Deposition of Eugene Martin, page 93). Those odors were more associated with hooking and unhooking hoses than they were with the fittings at the terminals (Deposition of Jimmy Melvin, page 63). Mr. Melvin never observed leakage from the terminal fittings (Deposition of Jimmy Melvin, page 63).

According to Mr. Melvin, Calzona/Coastal hauled more gasoline than diesel fuel and Mr. Webb would have hauled both (Deposition of Jimmy Melvin, pages 97-98). Mr. Webb would have hauled jet fuel as needed and he did not recall Mr. Webb hauling any avgas (Deposition of Jimmy Melvin, page 98).

According to Mr. Super, Mr. Webb hauled jet fuel on almost a daily basis between 1985 and the early 1990s (Deposition of Robert Super, page 35). During that time all jet fuel for Sky Harbor Airport had to be delivered by truck because there was no pipeline at that time (Deposition of Robert Super, page 33). Sometime in the early 1990s a pipeline was built between the Phoenix

Terminal and Sky Harbor Airport (Deposition of Robert Super, page 34). Mr. Super stated that if everything went right, they could deliver three loads of jet fuel during their shift (Deposition of Robert Super, page 34).

There was no avgas in the Phoenix area (Deposition of Robert Super, page 35). Sometimes drivers brought the avgas form the West Coast and since they were out of hours, Calzona/Coastal drivers, including Mr. Super and Mr. Webb, delivered this fuel to Sky Harbor Airport and smaller airports (Deposition of Robert Super, pages 35-46).

Occasionally, Calzona/Coastal would be contacted to pump out a storage tank (Deposition of Robert Super, page 97). That may occur if the wrong fuel product was placed in the wrong tank (Deposition of Robert Super, page 97). This request was made approximately once per month or once every two months (Deposition of Robert Super, page 98). This process took two and a half to three hours and may have involved two trucks, depending on the tank's capacity (Deposition of Robert Super, page 100).

Mr. Super could not provide any estimate of what percentage of his time he spent at any given terminal and noted that was the same for Mr. Webb (Deposition of Robert Super, pages 266-267).

Mr. Webb's Training

Department of Transportation (DOT) Training

The DOT provides regulations that govern tanker operations hauling hazardous materials. [21] More specifically, for Mr. Webb to drive tanker trucks hauling flammable and combustible liquids such as gasoline and other fuels, he must have been tested on his knowledge and skills for the commercial motor vehicle license for Group A. This would include passing the written DOT exam; passing the driver's road test; and knowing how to safely load and properly block, brace, and secure the cargo.

In order to haul hazardous materials. Mr. Webb must obtain a state-issued endorsement for tank vehicles which requires a knowledge test followed by obtaining a hazardous materials endorsement. To do so, he must have knowledge of information contained in 49 CFR Parts 171, 172, 173, 177, 178, and 397 on including:

- Hazardous materials regulations;
- Hazardous materials handling;
- Operation of emergency equipment; and,
- Emergency response procedures.

More specifically, 49 CFR Part 177 addresses transportation on a public highway and Part 177.800 addresses the responsibility for compliance and training. This section stated that each

carrier/employer shall comply with all the applicable requirements in this part and shall ensure its hazmat employees receive training in relation thereto. It also stated that a carrier/employer may not transport a hazardous material by motor vehicle unless each of its hazmat employees involved in that transportation is trained as required by this part and subpart H of part 172. Subpart H of Part 172 requires the hazmat employee to be trained in the following areas:

- To recognize and identify hazardous materials consistent with the hazard communication standards;
- Function-specific training applicable to the functions the employee performs;
- Emergency response information;
- Measures to protect himself from the hazards associated with the hazardous materials he may be exposed to in the workplace including specific measures the employer has implemented to protect employees against exposure;
- Methods and procedures to avoid accidents;
- Security awareness training; and
- Training conducted by employers as required for compliance with the hazard communication standards required by OSHA (29 CFR 1910.120 or 1910.1200). Training should be provided initially, when job function changes, and every three years thereafter. Each hazmat employer is responsible for compliance with these requirements.

Coastal Transport is known as a hazmat carrier because they engaged in the transportation of hazardous materials (Deposition of Barry Detlefsen, page 38). Hazardous materials are products that the secretary of transportation has deemed pose an unreasonable risk to the health, safety, and property while engaged in commerce (Deposition of Barry Detlefsen, page 38). Therefore, they had to abide by those regulations, including the training of hazmat employees, including Mr. Webb (Deposition of Barry Detlefsen, page 38). Hazardous Material Regulations 172.700 specified the type of training that must be presented to the hazmat employees (Deposition of Barry Detlefsen, page 38). Training must include awareness of hazardous materials and function-specific training (Deposition of Barry Detlefsen, page 39). The hazardous waste awareness training included identification of hazardous material, the proper preparation of shipping papers, placarding and marking the vehicle, and handling of hazardous materials using proper personal protective equipment (Deposition of Barry Detlefsen, page 40).

As drivers for Calzona/Coastal, Mr. Super and Mr. Webb had a commercial driver's license and held a HazMat endorsement (Deposition of Robert Super, pages 76-77). They received hazardous material training while employed by Calzona/Coastal (Deposition of Robert Super, page 77).

Coastal had to train, test, and certify their hazmat employees (Deposition of Barry Detlefsen, page 92). That was part of the training required by 49 CFR 172.700 (Deposition of Barry Detlefsen, page 92). Mr. Detlefsen's role with Coastal was to make sure that the training they offered met DOT regulatory standards and that every hazmat employee attended and completed

the training (Deposition of Barry Detlefsen, page 93). Follow-up training was required every 36-months (Deposition of Barry Detlefsen, page 93).

Mr. Webb's DOT road and written tests were completed on 25 January 1985. [22] Mr. Webb's commercial driver's license was renewed on 27 December 1991 and he had a tank/hazardous material combination endorsement at that time. [23]

Calzona/Coastal Training

Calzona and Coastal had driver training manuals and Policies and Procedures for Driver Personnel (Deposition of Robert Super, page 46; Deposition of Jimmy Melvin, page 69; Deposition of Eugene Martin, page 40). [24] [25] The Calzona and Coastal training manuals contained essentially the same information and the purpose of this manual was to provide drivers with information about how to perform their job safely (Deposition of Robert Super, page 46; Deposition of Jimmy Melvin, page 69; Deposition of Eugene Martin, page 40). The drivers were taught that safety took precedence over expediency, hurrying up, and shortcuts (Deposition of Robert Super, page 59).

Coastal trained its employees on how to identify hazards of the materials, how to use protective measures, how to avoid accidents, and how to respond to an emergency when loading transporting and delivering motor fuels (Deposition of Barry Detlefsen, page 170). As part of the hazardous material training they received from Coastal, drivers learned about loading, handling, storage, transportation, and emergency response procedures (Deposition of Eugene Martin, page 41). Their training focused on the safe methods of loading and unloading products so that the driver or the public did not come into contact with liquids or vapor (Deposition of Jimmy Melvin, page 71; Deposition of Eugene Martin, page 28). Following this training drivers were required to take a test (Deposition of Eugene Martin, page 41).

According to Mr. Super, both Calzona and Coastal had training coordinators, whose job it was to show new drivers how to perform their job including loading and unloading liquid products (Deposition of Robert Super, page 49; Deposition of Jimmy Melvin, page 81). The training coordinators had to have knowledge of the equipment that was to be used and the products they were delivering (Deposition of Robert Super, page 50). Mr. Webb, Mr. Super and Mr. Martin served as training coordinators when they were employed with Coastal (Deposition of Robert Super, page 50; Deposition of Jimmy Melvin, page 80; Deposition of Eugene Martin, page 159).

Drivers were taught to avoid contact with any liquid products that they hauled and to prevent inhaling vapors from those products (Deposition of Robert Super, pages 53; 107). Drivers had to carry in their trucks the USDOT Hazardous Material Regulations, the USDOT Federal Motor Carrier Safety Regulations and the USDOT Emergency Response Guidebook (Deposition of Robert Super, page 54; Deposition of Eugene Martin, page 136). Copies of the Driver Policy Manual and Driver Training Manual were kept in each employee's locker (Deposition of Robert Super, page 54).

Calzona/Coastal had safety meetings that were held about every month (Deposition of Robert Super, page 60; Deposition of Barry Detlefsen, page 76) or at least every six months (Deposition of Jimmy Melvin, page 72) or about once per year (Deposition of Eugene Martin, pages 131; 169). During that time, they discussed any safety infractions that were observed or complaints that had been received from terminals and how to load and unload product safely and properly (Deposition of Robert Super, page 60; Deposition of Jimmy Melvin, page 72; Deposition of Eugene Martin, page 132).

Calzona/Coastal drivers were taught and encouraged to report any unsafe conditions they observed at a loading rack and knew that they would not be fired for doing so (Deposition of Robert Super, page 66; Deposition of Jimmy Melvin, pages 73; 108; Deposition of Eugene Martin, page 133).

Drivers were required to watch safety videos at Calzona/Coastal (Deposition of Jimmy Melvin, page 89). Mr. Melvin did not recall if the videos included information on loading and unloading products, he did recall watching a video that discussed how to read a material safety data sheet (MSDS) (Deposition of Jimmy Melvin, page 89).

Coastal trained their drivers about the different grades of gasoline (Deposition of Barry Detlefsen, page 115). Coastal provided its drivers with MSDS regarding the product they were transporting (Deposition of Barry Detlefsen, page 115). Coastal was responsible to make sure their drivers were informed about the dangers of handling the products (Deposition of Barry Detlefsen, page 115).

Drivers at Calzona/Coastal received training on MSDS and how to read them (Deposition of Robert Super, page 125; Deposition of Barry Detlefsen, page 116). They were taught that MSDS contained information about the product they were hauling and the health risks (Deposition of Robert Super, page 125). Drivers were required to carry the MSDS for the product they were hauling (Deposition of Robert Super, page 89; Deposition of Jimmy Melvin, page 36). However, Mr. Martin disagreed and said that MSDS for products they were hauling were not in the trucks (Deposition of Eugene Martin, page 118).

Mr. Super testified that Calzona/Coastal trained him on how to protect himself from the hazards associated with the materials he worked with and on the methods and procedures to avoid accidents or exposures (Deposition of Robert Super, page 82). Additionally, the drivers were trained on what to do in case of a spill or other emergency (Deposition of Robert Super, page 87).

When dealing with a hazardous material emergency, they had the bill of lading which contained the name of the hazardous materials they were hauling and, as previously stated, they were required to have the Emergency Response Guidebook (Deposition of Robert Super, pages 87-88; Deposition of Jimmy Melvin, page 77). This guidebook contained information about the hazards of different chemicals and what to do in case of a spill (Deposition of Robert Super, page 88).

According to Mr. Super, he felt that Calzona/Coastal provided him with adequate instructions on how to perform his job and how to perform his job safely (Deposition of Robert Super, page 249).

Mr. Webb received "*First Responder Awareness Level Training*" on 26 April 1991. [26] This training was designed to provide information on what hazardous materials were and the risks associated with them during an incident, the potential outcomes associated with an emergency when hazardous materials were present, the ability to recognize and identify hazardous materials, the role of the first responder, and the ability to realize the need for additional resources.

In 1992, Mr. Webb completed a 16-hour training course on hazardous waste transportation. [27] This course covered information on product handling, documentation, labeling and placarding, loading and handling, incident reporting/emergency procedures, state and federal regulations, safe operation of vehicles, and inspection of vehicles and containers.

In August 1998, February 2001, January 2004, January 2007, January 2010 and February 2013, Mr. Webb completed recertification training courses in safe handling and transportation of hazardous materials. [28] [29] [30] [31] [32] [33] As part of these courses he completed a written test and acknowledged the risks associated with the transportation of petroleum products.

Loading Rack Training

Prior to being able to load at a fuel terminal and receiving their loading card, a driver had to demonstrate to the yard manager or terminal manager that they could perform the precise loading procedures (Deposition of Robert Super, page 39; Deposition of Jimmy Melvin, pages 28; 81). This process was repeated at each terminal for which the driver needed to load and the policies and procedures were consistent from terminal to terminal (Deposition of Robert Super, pages 39; 111; Deposition of Jimmy Melvin, page 81). If a carded driver made a mistake when loading at a rack, they could be banned from loading at that terminal (Deposition of Robert Super, page 39; Deposition of Jimmy Melvin, pages 81-82; Deposition of Eugene Martin, page 174). The drivers knew that they had to follow the policies and procedures not only at the rack, but also when driving and unloading (Deposition of Robert Super, page 41; Deposition of Eugene Martin, page 90). If a yardman or other employee at a loading terminal observed a driver not following the rules they would "*... call you out ...*" (Deposition of Robert Super, page 164).

At the loading racks, terminals had signs that contained the procedures for loading including grounding the vehicle, hooking up vapor recovery hose, etc. (Deposition of Robert Super, page 55). These signs also warned the drivers to avoid skin contact or inhalation of products and to report spills, no matter how small (Deposition of Robert Super, pages 55-56). If there was a leak or spill, they were instructed to shut down the rack immediately (Deposition of Robert Super, page 56; Deposition of Eugene Martin, page 192). Once they received the loading card, drivers were also provided with written rules and regulations for the loading facility (Deposition of Eugene Martin, page 185).

Mr. Melvin saw signs posted (location not stated) which warned about gasoline being hazardous to your health and noted that Mr. Webb would have seen those signs (Deposition of Jimmy Melvin, page 100). The warnings were not for benzene as there was no benzene-specific concern, but rather for the gasoline as the total hydrocarbon.

Mr. Super acknowledged that, based on the rules and oversight he observed, the terminal owners and operators where making sure the fuel loading systems were used properly (Deposition of Robert Super, page 112). These owners and operators were concerned about making sure that the system stayed vapor tight and that there were no leaks or spills (Deposition of Robert Super, page 113).

The bills of lading that they received from the terminals, specifically the Kinder Morgan/SFPP terminal, indicated that MSDS were available in the terminal offices (Deposition of Jimmy Melvin, page 37). Mr. Martin agreed that MSDS were available for review at the terminals (Deposition of Eugene Martin, page 118).

Fuel Product Terminal Overview

The loading racks where Mr. Webb loaded fuel products had similar training requirements prior to being allowed to load at the individual facilities and similar loading procedures. The loading racks had agreements with the trucking companies that tank truck drivers had been trained in the handling and transportation of hazardous materials and, prior to receiving their loading cards, drivers were required to be trained and documented that they were able to safely load fuel products at the terminals.

The fuel loading procedures were similar and involved making sure that there were no spills prior to loading, hooking up ground cables, vapor recovery lines, and fuel dispensing lines, and standing near the loading meter in case of an emergency, and then picking up their bill of lading before exiting the terminal. Drivers were required to report any leaks or drips they observed to the terminal management.

MSDS for the fuel products that were loaded were available upon request at each terminal.

All the facilities conducted daily, monthly, and annual inspections and any leaks or spills were reported and repaired. Some facilities have conducted air monitoring of their employees during work tasks. Summarized below is information about loading procedures, inspections, and available air monitoring data and MSDS for the ConocoPhillips, Kinder Morgan/SFPP, Caljet, Pro Petroleum, and Circle K loading racks and Valero fuels.

As previously stated, OSHA recognized that potential benzene exposures at fuel distribution terminals that had vapor recovery systems were substantially below the OSHA benzene action level of 0.5 ppm. [20] "*Thus OSHA concluded that the use of either type of vapor control system would result in average exposures virtually always below the action level and proposed to*

exempt from this action loading operations at both bulk plants and terminals which use the vapor control systems on this basis."

ConocoPhillips (Tosco) Loading Rack

Coworker Testimony about Mr. Webb's Work at the ConocoPhillips (Tosco) Loading Rack

According to Mr. Super, Mr. Webb loaded gasoline products at this facility between 1985 and 2002 (Deposition of Robert Super, page 153). Mr. Super did not know if Mr. Webb loaded at this facility on a regular basis (Deposition of Robert Super, page 154). Furthermore, Mr. Melvin commented that the Mr. Webb would have loaded fuel at this facility between 1984 and 1995, but he could not say how frequently that occurred (Deposition of Jimmy Melvin, page 50). Below I refer to this facility as "*Tosco*" or "*ConocoPhillips,*" but it is all the same location.

No information was available about the frequency or type of fuel products Mr. Webb loaded at the Tosco terminal during its ownership and operation between 1997 and 2008.

Facility Information

Tosco Corporation purchased a terminal located at S. 51st Ave., Phoenix, AZ 85043 from Union Oil Company of California in December 1996 and began operating it in 1997. [34] Phillips Petroleum Company acquired the terminal after merging with Tosco in 2001. [34] ConocoPhillips then acquired this asset as part of a merger between Conoco Inc. and Phillips Petroleum Company in late 2002. [34] This asset was sold by ConocoPhillips to a Kinder Morgan/SFPP affiliate in December 2008 (Deposition of Dennis Gilmore, page 50).

Douglas Kleopfer was employed at Tosco's Phoenix terminal from late 1989 until 1990 (under the ownership of Unocal) as the terminal foreman and returned in 1999 as the terminal's superintendent (Deposition of Douglas Kleopfer, pages 15-16). [35] The only changes between 1990 and 1999 was that in 1990 they may have been using Chevron's vapor recovery and by the time he returned in 1999 Tosco had their own vapor recovery unit (Deposition of Douglas Kleopfer, page 18). He noted that he may have been confusing that terminal with another (Deposition of Douglas Kleopfer, page 18). Top loading was not occurring in 1990 at the former Unocal facility (Deposition of Douglas Kleopfer, page 19).

As of 1990 the Unocal facility received gasoline and diesel from the Kinder Morgan/SFPP pipeline and trucks were loaded to deliver those products to the 76 branded stations in the Phoenix area (Deposition of Douglas Kleopfer, page 26).

Dennis Gilmore was employed by ConocoPhillips at the Phoenix terminal from 2001 to 2008 as the terminal supervisor (Deposition of Dennis Gilmore, page 10). [36] Mr. Gilmore began his employment with Union Oil in 1971 as a tank truck driver (Deposition of Dennis Gilmore, page

10). Since then he worked as a terminal supervisor at various terminal and pipeline facilities (Deposition of Dennis Gilmore, page 10).

The ConocoPhillips facility had five employees at the facility and it was manned 16 hours per day (Deposition of Dennis Gilmore, page 16). There was always a person on call during off-hours (Deposition of Dennis Gilmore, page 16).

The Phoenix facility had three lanes for loading gasoline and one for diesel fuel (Deposition of Dennis Gilmore, page 54). However, diesel fuel could be loaded in the gasoline lanes (Deposition of Dennis Gilmore, page 54). The facility, through their mechanical systems and pumps, blended gasoline with additives before the final mixture was loaded into the tank trucks (Deposition of Dennis Gilmore, page 55).

The gasoline hose connections were dry so there should not be any vapors being emitted during connection or disconnection (Deposition of Dennis Gilmore, page 48). The vapor recovery collected vapors from the trucks during loading operations (Deposition of Dennis Gilmore, page 48). The vapors were returned to a unit that processed the vapors so they would not be emitted to the atmosphere (Deposition of Dennis Gilmore, page 55).

The vapor recovery system was designed so that if it went down the whole rack was shut down (Deposition of Dennis Gilmore, page 86). That was a regulatory requirement (Deposition of Dennis Gilmore, page 86). The vapor recovery system was automated so there was no way for a tank truck driver to load a truck without the vapor recovery system operating (Deposition of Dennis Gilmore, page 87).

The benzene content of Tosco gasoline as of 1988 was approximately 1.7%. [37]

Tosco had a benzene policy that recognized the OSHA benzene standard (29 CFR 1910.1028) had requirements for training employees, air and medical monitoring, signage, and personal protective equipment for those personnel working with or exposed to benzene and benzene containing products (>0.1% benzene). [38] As part of that policy, Tosco understood that bulk wholesale storage facilities that employed vapor control for all loading and unloading or transportation of such materials in intact containers and pipelines was exempt from those regulations.

Loading Policies and Procedures

In order to load at the ConocoPhillips facility, a carrier had to have a signed agreement with ConocoPhillips to load at the facility (Deposition of Dennis Gilmore, page 69). [39] [40] The carrier was supposed to train their employees at the ConocoPhillips facility so that they were familiar with the ConocoPhillips loading procedures (Deposition of Dennis Gilmore, page 69). Additionally, the carrier was required to provide their drivers with hazardous material training (Deposition of Dennis Gilmore, page 69).

Following their company training, a driver had to watch a driver orientation video that went over the safe loading and handling procedures for petroleum products and pass a written test (Deposition of Dennis Gilmore, page 70; 72). A ConocoPhillips operator then monitored their loading to make sure the driver followed the correct procedures (Deposition of Dennis Gilmore, page 70). Once they passed and signed the load rack and driver loading card agreement, they were given their driver number and allowed to load at the facility (Deposition of Dennis Gilmore, page 70). [41] [42]

Mr. Kleopfer testified that when he was a terminal foreman in 1990, he made sure that the contract drivers were following the policies and procedures at the terminal and, if they were shorthanded, would perform training of a driver on the rack (Deposition of Douglas Kleopfer, page 45).

The tank truck loading procedures at this facility were as follows: [43] [44]

- Before entering the loading rack, stop at the painted line and turn off all electrical devices.
- Proceed into the loading rack slowly and once the truck has been positioned, the engine is to be shut off and all electrical switches are off.
- Plug the grounding cables into the truck and trailer and ensure the grounding system shows a green light.
- Connect the vapor recovery hose to the truck and trailer.
- Connect the appropriate loading arm couplers to the truck and trailer.
- Place the identification tags in their slot to identify the specific product being loaded into each compartment.
- Swipe the access card through the card reader and enter the requested data.
- Confirm the capacity of all the compartments that are to be filled and set the desired product quantity on the Accuload Preset Meter.
- When loading, the driver must be in attendance at all times.
- After loading disconnect the loading arms and return them to their stored positions.
- Disconnect the vapor hoses and return them to their stored positions.
- Disconnect the ground plugs and return them to their receptacles.
- End the loading process on the card reader.
- Start truck and exit the loading area.
- Obtain the bill of lading from the designated printer.
- Any equipment failure or malfunctions were to be reported immediately to the Tosco Operator on duty at the facility.

Facility Inspections

Facility personnel performed daily, weekly, monthly, and annual inspections of the loading rack to make sure that everything was operating properly (Deposition of Dennis Gilmore, pages 45;

73). The air quality board came to the site and witnessed their source tests for the vapor recovery unit on an annual basis (Deposition of Dennis Gilmore, page 73). [45] The daily, weekly, and monthly inspection were performed to make sure that everything was functioning properly and to make sure there were no leaks or drips (Deposition of Dennis Gilmore, pages 46; 73). [46] [47] [48] The drivers were also responsible for reporting drips, smells, or anything that was out of the ordinary (Deposition of Dennis Gilmore, page 73).

They also had fugitive emissions monitoring at the truck rack (Deposition of Dennis Gilmore, page 73). In order to do this, they used a MiniRAE to monitor valves, flanges, fittings throughout the facility including the tank farm and truck rack (Deposition of Dennis Gilmore, page 78). [46] [49] This direct reading instrument provided real time air monitoring.

If leaks were discovered, they were repaired immediately, or the loading lane was shut down so no one could load until it was fixed (Deposition of Dennis Gilmore, pages 47; 83; Deposition of Douglas Kleopfer, page 54). If there was an issue in the middle of the night, the driver called the operator on duty and the operator told the driver to cone off the lane so no one could use it until it was repaired (Deposition of Dennis Gilmore, page 83). All operators were authorized to fix whatever needed to be fixed and they maintained a supply of spare parts (Deposition of Dennis Gilmore, page 84).

Air Monitoring

As of 1977, Tosco was investigating air sampling methodologies and noted that personal air sampling could be conducted with a personal sampling pump, drawing air through a charcoal tube, and then desorbed and analyzed using gas chromatography. [50] The author noted that Tosco's Rocky Flats Research Center could perform the analyses in accordance with NIOSH's air sampling and analytical procedures.

Tosco had an Industrial Hygiene Management Plan, whose purpose was "*... to recognize, evaluate and control potential occupational exposures of chemical and physical agents in the workplace and maintain appropriate records.*" [51] This state of the art plan outlined procedures for identifying jobs with potential exposures, assessing the work area, evaluating exposures, interpreting and reporting results, and controlling exposures that are unacceptably high.

When Mr. Kleopfer worked at the terminal in 1990 and when it was operated by Unocal, they had their own drivers and periodically the industrial hygiene department performed air monitoring on terminal personnel and drivers (Deposition of Douglas Kleopfer, page 42). He recalled that when he returned in 1999, the industrial hygiene department continued to perform air monitoring on terminal personnel, but he did not recall if they performed monitoring on drivers (Deposition of Douglas Kleopfer, page 42).

During Mr. Gilmore's time at the ConocoPhillips facility between 2001 and 2008 no air monitoring was performed on drivers (Deposition of Dennis Gilmore, page 22). However, air monitoring was performed on terminal employees during their maintenance tasks at the truck rack, tank farm, and while they worked in the lab or when gauging tanks (Deposition of Dennis Gilmore, pages 23; 91). Mr. Gilmore stated that he felt that a typical truck driver would spend approximately an hour to an hour and a half at the loading rack and that an operator typically spent approximately two hours around the loading rack (Deposition of Dennis Gilmore, page 94). Therefore, the benzene exposure for the driver would be similar to the operator (Deposition of Dennis Gilmore, page 94).

I had the opportunity to speak with Tom Thompson, a former Unocal, Arco, and Tosco industrial hygienist who worked for those entities from beginning in 1989. Mr. Thompson stated that he has performed sampling at the Tosco facility when it was owned by Unocal. Air sampling was performed on an annual basis by himself (70%) or a contractor (30%) and they used an American Industrial Hygiene Association (AIHA) accredited laboratory to perform the analysis. Mr. Thompson did not recall any personal sampling data that approached the OSHA action level for benzene of 0.5 ppm and most samples were below the limit of detection (limit of detection not stated).

The available sampling data was collected in 2001, 2005, and 2008 on Tosco operators. [52] [53] [54] [55] A 11 December 2001 personal sample collected on an operator reported benzene 8-hour time weighted average concentrations below the limit of detection (<0.021 ppm). [54] During that time the operator spent four hours outside performing maintenance on the loading rack, took gasoline samples (3 minutes) and spend the remainder of the time indoors doing paper work. [54] Four additional personal samples collected on 10 and 11 December 2005 and 30 January 2008 reported 8-hour time weighted average benzene concentrations that ranged from 0.014 ppm to 0.018 ppm. [55] [52] No information was available regarding the work tasks these individuals performed during those sampling times.

ConocoPhillips Material Safety Data Sheets

I have reviewed Phillips 66 MSDS for gasoline dated 26 February 1999 and 11 May 2001 as well as Unbranded gasoline dated 26 September 2002 and 1 January 2003. [56] [57] [58] [59] These MSDS were completely adequate with regard to the content as required by the OSHA Hazard Communication Standard (29 CFR 1910.1200). The recommendations provided the language necessary for the downstream employers to take appropriate protective measures, and implement procedures and processes to ensure a safe working environment.

Kinder Morgan/SFPP Loading Rack

Coworker Testimony about Mr. Webb's Work at the Kinder Morgan/SFPP Loading Rack

Mr. Super stated that the drivers received training on how to safely load fuel products at the Kinder Morgan/SFPP racks (Deposition of Robert Super, page 269). Based on his experience as a truck driver, he felt that the Kinder Morgan/SFPP facility was safe (Deposition of Robert Super, page 267). Additionally, Mr. Melvin commented that Mr. Webb would have loaded fuel at the Kinder Morgan/SFPP racks between 1984 and 1995 (Deposition of Jimmy Melvin, page 51).

Some information was available about the frequency and type of fuel products Mr. Webb loaded at the Kinder Morgan/SFPP terminal during its ownership and operation. [60]

Facility Information

Vapor recovery manufacturing specification were developed by HydroTech Engineering for the Kinder Morgan/SFPP facility in August 1979. [61] Site drawings dated 1979/1980 documented that the drawings were put out for bid in May 1980 and construction drawing were released in June 1980. [62] Construction photographs documented that the vapor recovery facility was being constructed in the fall of 1980 and the Kinder Morgan/SFPP vapor recovery equipment was operational by the end of 1980 or early 1981. [62]

Loading Policies and Procedures

Casey Alleman began work at Kinder Morgan/SFPP in 2010, starting as an operations supervisor and is currently an area manager (Deposition of Casey Alleman, page 8). [63] He is currently the ultimate decision maker at the Phoenix terminal (Deposition of Casey Alleman, page 9). All drivers were required to participate in a Kinder Morgan/SFPP driver safety class before loading unobserved and they were supposed to repeat that class every three years (Deposition of Casey Alleman, page 9). Exhibits 1 and 2 to Mr. Alleman's deposition are Kinder Morgan/SFPP class rosters from 2009 and 2012 that documented Mr. Webb's his attendance at those classes. [64]

In order to receive a Kinder Morgan/SFPP loading card, drivers had to have a hazmat endorsement, attend the driver safety class, they had to load with a training driver at the carrier that employed them, they had to sign a low sulfur training affidavit, and they had to review the Phoenix evacuation plan (Deposition of Casey Alleman, page 14). [65] Kinder Morgan/SFPP maintained driver safety class records for five years (Deposition of Casey Alleman, page 15).

Kinder Morgan/SFPP did not provide benzene specific training, but their customers hired hazmat carriers and the expectation and understanding was that they were aware of the products potential hazards when loaded on their trailers (Deposition of Casey Alleman, pages 15-16).

Kinder Morgan had tank truck loading and unloading safety rules and procedures for truck drivers and employees. [66] [67] [68] [69] The tank truck loading procedures at this facility were as follows:

- As soon as the truck is spotted, engine switch, lights, and all radios and cellular phones must be turned off and doors closed. No truck was to be parked or stopped with engine running, radio, phone or lights on or doors opened near loading rack.
- Insert the driver card into the reader and enter the requested information.
- Connect ground-high level shut off device to the truck and/or trailer being loaded.
- Properly connect vapor recovery hose to the truck and/or trailer being loaded, ensuring that the coupler is correctly seated and the dog-ears are completely clamped down.
- Select product and enter gallons desired on touch screen.
- Push start button and begin loading. Check vapor hose, product hoses and all connections for vapor emissions or product leaks. If leaks are found, shut down loading and call the terminal Operator to report problem/request repairs.
- The truck must be attended during the entire loading process. Driver must stand by set-stop counter or meter display unit while loading is in progress and remain at that station until loading is complete.
- Trucks were not to be restarted and re-spotted unless loading arms, vapor hoses, high level shut off device and grounds have been disconnected in listed order and placed in their storage position.
- In the event of a spill, vapor leak, liquid leak, or any other leak, drivers were to cease loading operations and contact the terminal Operator immediately for clean-up procedures. No trucks should enter the rack or pass close to the spill until clean-up is completed.
- After loading is completed, close loading valve, and remove loading arm, vapor hose, high level shut off device and grounds in the order listed and return them to their storage position. Ensure that all vapor hoses are returned to their hanger.
- Remove loading card from card reader.
- Remove bill of lading from the ticket printer.

Facility Inspections

Kinder Morgan/SFPP personnel perform rack inspections twice per day and they did monthly inspections (Deposition of Casey Alleman, page 29). [70] When they look for leaks in a rack, they perform what they call sight, sound, and smell inspection and forms are filled out (Deposition of Casey Alleman, page 29). [70] They actively looked for leaks around the facility (Deposition of Casey Alleman, page 30). Mr. Alleman has not detected an odor of gasoline around the facility (Deposition of Casey Alleman, page 30). If they did smell gasoline, something was not functioning properly and it was their procedure to stop loading and make the appropriate repair (Deposition of Casey Alleman, page 31). [71] The odor threshold for gasoline is 0.25 ppm for gasoline as compared to the TLV which is 300 ppm as an 8-hour TWA. [12]

In addition, Maricopa County Air Quality Department inspectors performed permit compliance and vapor recovery equipment safety inspections that consistently found Kinder Morgan in compliance and exceeding recovery efficiency standards. [65]

Air Monitoring

Air monitoring was conducted at the Kinder Morgan/SFPP facility in 1991, 1999, 2015, and 2019. [72] [73] [74] [75] In 1991 air sampling was conducted for benzene and other constituents on an operator (103 minutes) and on a laboratory technician (89 minutes). [72] The report noted that the operator spent much of their time in the control house monitoring pipeline flow and that the laboratory technician performed routine laboratory work, which included work under a laboratory hood. The reported benzene concentrations were below the limit of detection, which for the operator was less than (<) 1 ppm and for the laboratory technician was less than (<) 3 ppm.

Personal air samples were collected and analyzed for benzene and other constituents on 15 March 1999 at the Kinder Morgan/SFPP Phoenix Terminal. [73] Four personal samples were collected on two operators. Samples were collected and analyzed in accordance with OSHA Method 7 and analyzed by an AIHA accredited laboratory. During the sampling period, operators performed a variety of gauging, maintenance, and sampling tasks. All benzene sampling results were below the limit of detection, which ranged from less than (<) 0.01 ppm to less than (<) 0.02 ppm.

Air sampling for benzene was conducted at the Kinder Morgan/SFPP Phoenix Terminal between 15 and 17 June 2015. [74] During that time ten area samples were collected in loading racks for both full shift and short term concentrations and ten personal samples were collected for full shift concentrations and one personal sample was collected for short term concentrations. Samples were collected and analyzed in accordance with NIOSH Method 1500/1501 by an AIHA accredited laboratory. Specific information regarding what tasks were being conducted by terminal personnel during the sampling process was not provided. All benzene concentrations were below the limit of detection which ranged from less than (<) 0.09 ppm to less than (<) 0.2 ppm.

Air sampling was conducted on 11 and 12 July 2019 during the fuel loading process at the Kinder Morgan/SFPP, Phoenix Terminal. [75] A total of 15 air samples were collected and analyzed in accordance with NIOSH Method 1501 by an AIHA accredited laboratory. Two personal air samples were collected on an operator that shadowed the driver at each loading rack from the time that the driver got out of the truck, during the loading process, and until he got back in the truck to leave the facility. Sampling time ranged from 14 to 23 minutes and all benzene concentrations were below the limit of detection that ranged from less than (<) 0.133 ppm to less than (<) 0.224 ppm.

Caljet Loading Rack

Coworker Testimony about Mr. Webb's Work at the Caljet Loading Rack

According to Mr. Super, Caljet operated more than one loading rack in the Phoenix area (Deposition of Robert Super, page 156). Between 1985 and 2002, Mr. Super testified that Mr. Webb loaded gasoline products at Caljet's loading rack (Deposition of Robert Super, page 156). Mr. Super did not know if Mr. Webb loaded at these facilities on a regular basis (Deposition of Robert Super, page 157).

According to Mr. Martin, Caljet was the only loading rack that had top loading equipment without vapor recovery for gasoline (Deposition of Eugene Martin, page 97). Mr. Martin stated that the top loading process was discontinued at Caljet at some point before 2000 (Deposition of Eugene Martin, pages 97; 115). However, Mr. Millican, Caljet's former Director of Operations, indicated that he was not aware of any top-loading of gasoline at the Phoenix Tank Farm by the mid-1980s (Deposition of John Millican, pages 25-26). Mr. Martin was unable to provide an estimate of how frequently Mr. Webb reportedly top loaded gasoline without vapor recovery at this location, if at all (Deposition of Eugene Martin, page 98).

No information was available about the frequency or type of fuel products Mr. Webb loaded at the Caljet terminal during its ownership and operation.

Facility Information

The Caljet terminals function as a public warehouse, allowing gasoline owned by others to be stored at Caljet facilities, and then made available to various customers upon receipt at the terminals (Caljet's Fifth Supplemental Disclosure Statement, page 4; Deposition of Michael Gray, page 51; Deposition of John Millican, pages 84-85). [76]

Caljet of America, LLC operates three terminals within the Phoenix tank farm, each of which utilizes bottom loading technology and vapor recovery systems (Caljet's Fifth Supplemental Disclosure Statement, pages 3-4; Deposition of Michael Gray, pages 14, 60). [76] Caljet or its predecessors have operated Caljet's 53rd avenue terminal since approximately 1987 and its Monroe I and Monroe II terminals since approximately 1996 (Caljet's Fifth Supplemental Disclosure Statement, pages 3-4). [76] With regard to gasoline, Caljet's Monroe I and Monroe II terminals have been bottom loading terminals the entire time those terminals have been owned or operated by Caljet entities, and Caljet's 53rd Avenue terminal was a bottom loading terminal as of 1985 (Caljet's Fifth Supplemental Disclosure Statement, page 4). [76]

Loading Policies and Procedures

John Millican was a Caljet employee from 1995 until 2016 (Deposition of John Millican, page 8). [77] He was initially hired in 1995 as an operator and later became the terminal manager and

director of operations (Deposition of John Millican, pages 33; 91). Regarding the loading rack, it was Mr. Millican's belief that Caljet's goals were to safely and efficiently load the fuel products onto trucks (Deposition of John Millican, pages 56-57). The training he received was that gasoline was a hazardous material and to avoid exposure (Deposition of John Millican, page 62). Caljet operators train the drivers to follow the Caljet procedures and witness them performing those procedures (Deposition of John Millican, page 87). Once the driver can properly load three times following those procedures, that driver was provided with a Caljet loading card (Deposition of John Millican, page 87). Fuel truck drivers using Caljet's facilities are expected to report any leaks or spills so that loading can be discontinued until the issue is addressed (Deposition of Michael Gray, page 40).

The tank truck loading procedures at this facility were as follows: [78]

- Before entering the loading rack, stop at the yellow line and turn off all electrical devices. Only the truck motor can be operating.
- Drive under the loading rack and turn off the engine.
- Card in and enter the appropriate information on the computer.
- Hook up the ground cord and the vapor hose. There should be one green light and one orange light lit.
- Open internal valves and remove transportation caps.
- Make sure compartments are empty and loading heads are clean and clear of debris.
- Load all products needed.
- Driver must be present at the meter shutdown device at all times during the loading process.
- Close internal valves before disconnecting load arm.
- The load arm, vapor hose, and cord should be removed in that order.
- Remove tank truck from loading rack and proceed to the bill of lading room to card in and sign for the bill of lading.

Caljet had access to or was provided with Valero MSDS (Deposition of John Millican, page 108). Furthermore, MSDS for Valero's gasoline and all other gasoline stored at the terminals were available to anyone at the terminals that requested one, including drivers (Deposition of John Millican, page 108; Caljet's Fifth Supplemental Disclosure Statement, page 5). [78]

Facility Inspections

Caljet performed daily, weekly, monthly, and annual inspections of its equipment and replaced worn equipment, checked for leaks, and used daily sight, sound, and smell tests to observe if anything needed to be replaced or addressed (Deposition of Michael Gray, page 15). [79] [80] If a leak was detected it was repaired immediately (Deposition of John Millican, pages 92-93).

Air Monitoring

Air monitoring was conducted on 27 March 2012 on a Caljet laboratory employee for benzene. [81] Sampling and analysis was conducted in accordance with OSHA Method 7 by an AIHA accredited laboratory. The 8-hour TWA benzene concentrations were reported to be below the limit of detection (<) 0.033 ppm.

Air monitoring was conducted by ATC at the Caljet facility on 11 July 2019 in order to assess benzene exposures. [82] The purpose of this assessment was to assess potential exposures to benzene while loading the fuel trucks. The report noted that each driver typically spends 15 to 25 minutes at the station while loading, however 8-hour samples were collected in these areas. Area air samples were collected near the driver work station location midcenter on the loading rack lanes. Samples were collected over an 8-hour period using passive monitoring badges and analyzed in accordance with NIOSH Method 1501 by an AIHA accredited laboratory.

A total of eight, shift long area samples were collected, three at the 53rd Avenue terminal, three at Monroe 1 and two at Monroe 2. All samples were below the limit of detection (<) 0.1 ppm. According to the report, employees noted that the workload was normal during the assessment time period.

Pro Petroleum Loading Rack

Coworker Testimony about Mr. Webb's Work at the Pro Petroleum Loading Rack

Between the 1990s and 2002, Mr. Super testified that Mr. Webb loaded jet fuel and diesel fuel at the Pro Petroleum rack (Deposition of Robert Super, pages 168; 261). This facility did not have gasoline and mainly had jet fuel and diesel fuel (Deposition of Robert Super, pages 168; 261). Mr. Super observed Mr. Webb loading fuel product on approximately six occasions but Mr. Webb could have been there when Mr. Super was not (Deposition of Robert Super, pages 262-263). He did not know if Mr. Webb loaded diesel or jet fuel (Deposition of Robert Super, page 263).

When Mr. Martin began working in 1997, the Pro Petroleum rack did not have vapor recovery for its diesel loading, but they eventually (time not stated) added vapor recovery (Deposition of Eugene Martin, page 103).

Mr. Melvin testified that Mr. Webb loaded at the Pro Petroleum rack and that Mr. Webb loaded more diesel than gasoline (Deposition of Jimmy Melvin, page 47). Additionally, Mr. Martin testified that the Pro Petroleum racks only dispensed diesel fuel and that they did not load there frequently, maybe once per week (Deposition of Eugene Martin, page 73; 221).

No information was available about the frequency or type of fuel products Mr. Webb loaded at the Pro Petroleum terminal during its ownership and operation.

Facility Information

Pro Petroleum operated a facility located at 408 S. 43rd Ave, Phoenix, AZ 85009 from 1989 to the present. [83] [84] Calzona/Coastal did little business with Pro Petroleum during the time Mr. Webb was working for them. [83] Additionally, contrary to the testimony of Mr. Melvin, Pro Petroleum was not a gasoline loading facility. [83] Instead, Pro Petroleum dealt only in diesel and bio-diesel fuels. [83] Contrary to the testimony of Mr. Martin, the facility utilized a bottom loading system, where the trucks were hooked up to a hose and vaper-collecting device prior to loading the fuel. [83]

Loading Policies and Procedures

Prior to receiving a card to load at the Pro Petroleum facility, driver training must be performed by certified drivers employed with the same company as the driver trainee unless alternate arrangements are made with terminal personnel. [85] The driver trainees must perform all loading procedures under the supervision of a certified driver or terminal operator on three separate occasions (loads) prior to loading the 4th load under the observation of the Pro Petroleum terminal/rack operator. [85]

The tank truck loading procedures at this facility were as follows: [85] [86] [87]

- Prior to entering the truck loading rack, all vehicles must come to a complete stop at the yellow stop line located at the entrance to the lane. Vehicle headlights and electrical devices other than the vehicle engine must be turned off
- All vehicle engines must be turned off, all vehicles must be properly parked and vehicle brakes, including the tank vehicle's brakes, must be properly engaged before the driver connects any loading equipment to the tank vehicle or begins any loading procedures. The vehicle engine must remain off until loading is complete and all loading equipment has been disconnected from the tank vehicle.
- The tank vehicle must be equipped with a Scully compatible overfill protection system and a Scully Ground Bolt. The lights just east of the loading arms on the rack indicate a proper connection. Drivers must hook up the vapor hose in proper sequence so as not to lose any vapors already contained in the tank vehicle. The driver must connect the correct loading lines to the tank vehicle compartments designated to receive product via the dry brake couplings.
- The truck loading rack automation system at the Pro Petroleum facility is a cardless system. In order to receive loading authorization, the driver must enter the required information.
- During the entire loading process, the driver must remain in attendance in front of the data entry box. This requirement enables the driver to read the preset display, observe loading connections and press the emergency shutdown switch in case of an emergency.

- If product overflows a compartment or an emergency arises, the driver must push the emergency shutdown switch located on either end of the rack, and contact terminal personnel immediately. In the event of a product spill, no vehicle engine may be started until the spill has been cleaned up completely using the water, hose and squeegee provided.
- After loading is complete, the driver must disconnect the loading equipment and safety equipment from the tank vehicle in the following order:
    - Loading lines (dry-brake couplings)
    - Vapor recovery hose
    - Overfill protection/grounding cable/plug
- Prior to exiting the truck loading rack, the driver must verify that all loading rack equipment has been disconnected from the tank vehicle.
- After exiting the rack, the driver must properly park the vehicle in front of the bill of lading shack. By the time the driver arrives in the shack, the printer should have printed a five copy Bill of Lading. The driver must verify the accuracy of all information on it and sign all five copies in the space provided.

When dealing with spills the Pro Petroleum rules required that in the event of a product spill on the truck loading/unloading rack, which is large enough, to wet the vehicle (including the tank vehicle) or the concrete pad, no vehicle engine may be started until the spill has been cleaned up completely using the water, hose and squeegee provided. [85] The person who caused the spill must clean up all spills immediately, completely, and in accordance with all applicable environmental regulations. [85] Spills, which extend beyond a truck loading/unloading rack containment area and spills of large quantity, which do not extend beyond a truck loading/unloading rack containment area must be reported to terminal personnel immediately. [85] Spill buckets must be used when hooking up or unhooking loading equipment. Drips on the concrete must be wiped up immediately. [85]

Facility Inspections

Based on Pro Petroleum document production, Pro Petroleum calibrated/inspected the following areas/equipment:

- Calibrated the combustible gas detection sensor on a quarterly basis. [88]
- Inspected the vapor combustion unit on a daily basis. [89]
- Tested the combustible gas detection sensor on a weekly basis. [90]
- Performed weekly leak detection and repair [91]
- Performed a weekly inspection of the loading racks. [92]

Information about when these inspections began was not provided.

Air Monitoring

Air monitoring was conducted by ATC at the Pro Petroleum facility on 17 July 2019 in order to assess benzene exposures during fuel truck loading. [93] The purpose of this assessment was to assess potential exposures to benzene while loading the fuel trucks. The report noted that each driver typically spends 15 to 25 minutes at the station while loading, however 8-hour samples were collected in these areas. Area air samples were collected near the driver work station location midcenter on the loading rack lanes. Samples were collected over an 8-hour period using passive monitoring badges and analyzed in accordance with NIOSH Method 1501 by an AIHA accredited laboratory.

A total of two, shift long area samples were collected. All samples were below the limit of detection (<) 0.1 ppm. According to the report, employees noted that the workload was normal during the assessment time period.

Pro Petroleum Material Safety Data Sheets

I have reviewed the MSDS for a diesel product dated 7 May 2015, a biodiesel product dated 27 May 2016, and a jet fuel product dated 1 June 2016 distributed from the Pro Petroleum terminal. [94] [95] [96] These MSDS were completely adequate with regard to the content as required by the OSHA Hazard Communication Standard (29 CFR 1910.1200). The recommendations provided the language necessary for the downstream employers to take appropriate protective measures, and implement procedures and processes to ensure a safe working environment.

Circle K Loading Rack

Coworker Testimony about Mr. Webb's Work at the Circle K Loading Rack

It was Mr. Super's understanding that Circle K did not have any terminals (Deposition of Robert Super, page 170). Instead, they called a carrier and asked them to pick up a product at some rack (Deposition of Robert Super, page 170). ARCO owned and operated a terminal facility inside the Phoenix terminal between at least 1985 until around 2001 when it was acquired by BP (Deposition of Robert Super, page 227). At some point after that Circle K purchased the facility from BP (Deposition of Robert Super, page 227).

According to Mr. Melvin, he did not think Mr. Webb loaded very frequently for Circle K between 1984 and 1995 (Deposition of Jimmy Melvin, page 45). It was not until Mr. Melvin left Coastal in 1995 that Circle K had their own fuel loading rack (Deposition of Jimmy Melvin, page 45). He could not say if Mr. Webb loaded at a Circle K terminal after 1995 (Deposition of Jimmy Melvin, page 46).

No information was available about the frequency or type of fuel products Mr. Webb loaded at the Circle K terminal during its ownership and operation.

Facility Information

Circle K became the owner of the gasoline terminal located at 5333 W. Van Buren, Phoenix, Arizona in February 2010. [97] Mr. Webb first began loading fuel at the Circle K Terminal in November 2010 and his last fuel loading at the Circle K Terminal was in July 2015. [97] He loaded fuel at the Circle K Terminal intermittently between November 2010 and July 2015 as set forth in Circle K's bills of lading [97]

At the time of the purchase, the Circle K Terminal had two loading racks as well as a vapory recovery unit that transported vapors via underground piping to the nearby Kinder Morgan/SFPP facility for treatment/disposal (Deposition of Scott Mitchell, page 98). [97] In 2011, Circle K installed two additional loading racks at the Circle K Terminal as well as an onsite vapor recovery unit to treat/dispose of vapors on its own property, all of which was pursuant to a permit issued by Maricopa County. [97] Accordingly, there was a vapor recovery unit that was used for all fuel loadings the entire time Mr. Webb was present at the Circle K Terminal. [97]

Scott Mitchell was with Circle K since 2011 and has worked as a nighttime technician which involved training new drivers, receiving fuel off the pipeline, and making sure drivers on site followed the policies and procedures (Deposition of Scott Mitchell, pages 7-9). [98] He became the terminal manager on 1 May 2013 and his job duties were to oversee the terminal operations and make sure the safety policies and procedures were followed (Deposition of Scott Mitchell, pages 8-10).

According to Mr. Mitchell, prior to the terminal's ownership by Circle K in 2010, the terminal was owned and operated by Arco or BP (Deposition of Scott Mitchell, page 45). At that time, Arco/BP had their own products and own trucks to haul and deliver Arco/BP fuel (Deposition of Scott Mitchell, page 45).

The Circle K facility obtained Valero gasoline and diesel from the Kinder Morgan/SFPP pipeline (Deposition of Scott Mitchell, pages 26-28). Valero provided Circle K with its MSDS (Deposition of Scott Mitchell, page 120). Those MSDS were available to Circle K personnel and contract drivers (Deposition of Scott Mitchell, page 122).

Loading Policies and Procedures

Circle K had procedures for fuel loading at the Circle K terminal. [99] The tank truck loading procedures at this facility were as follows:

- Approach the staging line and shut off motors, lights, cell phones and other electrical equipment.
- Pull onto the loading rack and shut off engine.
- Connect the Scully grounding cord to the truck and trailer and verify the connection by observing the Scully light.

- Connect the vapor recovery line and then attach the loading arms and lock in place.
- Turn the dial to the appropriate product name for each compartment.
- Insert the loading card into the remote-control unit (RCU) and enter the appropriate information.
- Start the pump and inspect the area for leaks. The driver was to remain at the RCU throughout the loading process.
- If a leak was spotted, the driver was to stop loading immediately. All spills were to be reported to the facility management.
- After loading was complete, remove the load arm, the vapor hose, and the Scully.
- Pull out of the loading rack and proceed to the "*Dog House*" to obtain the bill of lading.

At the Circle K facility, it takes approximately 20 minutes to load a truck with fuel (Deposition of Scott Mitchell, pages 72-73). At some facilities it takes longer because the pumps do not run as fast (Deposition of Scott Mitchell, page 73).

Since Mr. Mitchell has been at the facility in 2011 there have been no fuel spills (Deposition of Scott Mitchell, page 77). There may have been some drips but no spills (Deposition of Scott Mitchell, page 78). When walking around his facility there may be some stains but there would be no liquid, because if there was, that would mean there was an issue and it would have been dealt with immediately (Deposition of Scott Mitchell, page 79).

Air Monitoring

Air monitoring was conducted by ATC at the Circle K facility on 17 June 2019 in order to assess benzene exposures. [100] A total of four shift long area samples were collected and one shift long personal sample. Two area samples were collected near the loading arms and two were collected at the operator stations in the loading racks. The personal sample was collected on a Circle K employee who spent the majority of his shift working near the loading rack. Air samples were collected with passive monitoring badges and analyzed in accordance with NIOSH Method 1501 by and AIHA accredited laboratory.

All samples were below the limit of detection. The personal sample result was less than (<) 0.2 ppm and the area samples were less than (<) 0.1 ppm to (<) 0.2 ppm. According to the report, employees noted that the workload was normal during the assessment time period.

Valero Fuels

Coworker Testimony about Mr. Webb's Work with Valero

Valero was not a rack owner, but Valero fuel products could have been distributed by the various terminals (Deposition of Eugene Martin, page 114). According to Mr. Martin, he and Mr. Webb

loaded Valero-branded fuels at Kinder Morgan/SFPP racks (Deposition of Eugene Martin, page 78). No information on frequency with which Valero products were loaded was provided.

Valero Fuel

Valero was not incorporated until June 25, 1995 and was not licensed to do business in Arizona until April 2002 (Deposition of Melinda Farias, page 38). [101] Therefore, Mr. Webb could not have hauled any Valero fuel product until after 2002.

Coastal and Valero had a Transportation Services Agreement with and effective date of 1 January 2006. [102] This agreement required all drivers to receive necessary training programs, licensing and certification required for the performance of the Services including, but not limited to training in the handling and transportation of hazardous materials. Furthermore, this agreement required the carrier to comply with all signs and other rules and regulations issued by the designated terminals and that drivers must be trained in all safety and security requirement of the terminal.

Melinda Farias was the executive director of wholesale marketing for Valero and has been with Valero since 2002 (Deposition of Melinda Farias, page 8). [103] In Phoenix they distributed branded and unbranded fuel (Deposition of Melinda Farias, pages 9-10). Circle K is an unbranded contract that they supplied fuel to (Deposition of Melinda Farias, page 17). Valero also has a relationship with Caljet because Valero stored some of its product at their terminal (Deposition of Melinda Farias, page 20).

As previously stated, the Circle K facility obtained Valero gasoline and diesel from the Kinder Morgan/SFPP pipeline (Deposition of Scott Mitchell, pages 26-28). Valero provided Circle K with its MSDS (Deposition of Scott Mitchell, page 120). Those MSDS were available to Circle K personnel and contract drivers (Deposition of Scott Mitchell, page 122).

Matthew Hodges has worked for Valero as the director of regulatory affairs in the environmental department since 2002 (Deposition of Matthew Hodges, pages 7-8). [104] His focus is on federal environmental regulations (Deposition of Matthew Hodges, page 13). Valero kept track of the benzene content of its fuel by analyzing every batch of gasoline for benzene (Deposition of Matthew Hodges, pages 23-24). [105] In 2011 the benzene content of gasoline changed because of the Mobile Source Air Toxic regulations that went into effect (Deposition of Matthew Hodges, page 24). Those regulations set the maximum benzene content in gasoline to be 0.62% by volume on an annual basis, unless credits were applied and then the maximum benzene content could be 1.3% by volume on an annual basis (Deposition of Matthew Hodges, page 24).

Valero Material Safety Data Sheets

Valero has MSDS available on the internet for people that need them (Deposition of Melinda Farias, page 25). I have reviewed Valero and Diamond Shamrock MSDS for gasoline dated 01

January 2002, 28 November 2005, 9 February 2009, 5 January 2010, 28 July 2011, 23 May 2014 [106] [107] [108] [109] [110] [111] [112] These MSDS were completely adequate with regard to the content as required by the OSHA Hazard Communication Standard (29 CFR 1910.1200). The recommendations provided the language necessary for the downstream employers to take appropriate protective measures, and implement procedures and processes to ensure a safe working environment.

Applicable Regulations

During the period of Mr. Webb's employment as a fuel truck driver (1985-2015) there was readily available knowledge of the potential hazards of exposure to gasoline, diesel fuel and other hydrocarbon chemicals. Additionally, as summarized below, there were regulatory programs, policies, and procedures in place at that time governing those types of operations.

Employer Responsibility

Mr. Webb's employers, Calzona Tankways and Coastal Transportation Company, were responsible for ensuring his safety in the workplace. By 1948, all states at that time had enacted Workmen's Compensation Laws for the purpose of compensating workers for on-the-job injuries and illnesses. According to Henshaw (2007), it was also *"intended to drive greater recognition among employers of their responsibility for workplace health and safety."* [113] Also in addition to state run programs, safety and health organizations had been long established, such as the American Society of Safety Engineers (ASSE) in 1911, National Safety Council (NSC) in 1913, the American Conference of Industrial Hygienists (ACGIH) in 1938, and the American Industrial Hygiene Association (AIHA) in 1939. [113] These organizations communicated hazards to employers and workers and also provided methods for prevention of injuries and illnesses.

According to Henshaw (2007):

> *"Responsible employers recognized the common law practices and societies' expectation of employer responsibility and by and large acted in a reasonable manner to protect their employees from injuries and illnesses."* [113]

The Occupational Safety and Health Act in 1970 defined the duty of an employer under the OSHA regulations is to provide a safe and healthful workplace. Specifically, these regulations defined the employer's responsibility to evaluate and convey the potential hazards associated with a product used by their employees and the measures necessary to protect the employees' safety and health. [114] Mr. Webb's employers had the responsibility to provide education and training regarding the safe work practices with the products, evaluate the potential exposures encountered by its employees, and ultimately were responsible for protecting the health of their employees. Employers must evaluate employee exposures and, as part of that evaluation, they must understand and consider the physical properties of a product, how those products are used

(tasks), the environment in which they are used (including ventilation), and the use of personal protective equipment.

Hazard Communication Standard

The purpose of the OSHA Hazard Communication Standard (29 CFR§1910.1200) was to establish uniform requirements for hazard communication in the manufacturing sector for worker health and safety. [115] Beginning in November 1985, OSHA's Hazard Communication Standard required chemical manufacturers and importers to evaluate the hazards of the chemicals they produced or imported, and to transmit this information to downstream employers by means of labels on containers and material safety data sheets. Furthermore, since May 1986 all covered employers, including the companies for whom Mr. Webb worked, were required to provide hazard information to their employees by means of labels on containers, Material Safety Data Sheets (MSDS), and training. [115] This program included teaching employees how to read, use and understand Material Safety Data Sheets (MSDS), where to find these documents at the site and how products should be labeled at the site.

Under the Hazard Communication Standard, gasoline is considered to be a mixture because it is a combination of two or more chemicals that is not the result of a chemical reaction. Furthermore, because gasoline has been tested as a mixture, the hazard communication standard requires manufacturers or importers to use that test data to determine its hazards. Therefore, it is not necessary to include a separate warning for benzene.

Drivers at Calzona/Coastal received training on MSDS and how to read them (Deposition of Robert Super, page 125; Deposition of Barry Detlefsen, page 116). They were taught that MSDS contained information about the products they were hauling and the health risks (Deposition of Robert Super, page 125). Drivers were required to carry the MSDS for the product they were hauling (Deposition of Robert Super, page 89; Deposition of Jimmy Melvin, page 36). However, Mr. Martin disagreed and said that MSDS for products they were hauling were not in the trucks (Deposition of Eugene Martin, page 118).

The bills of lading that they received from the terminals, specifically the Kinder Morgan/SFPP terminal, indicated that MSDS were available in the terminal offices (Deposition of Jimmy Melvin, page 37). Mr. Martin agreed that MSDS were available for review at the terminals (Deposition of Eugene Martin, page 118).

Under DOT regulations, there is no requirement for drivers to carry MSDS for the commodity they were carrying. Instead, they must carry shipping papers that include the basic description of the hazardous material including the Identification Number, the Proper Shipping Name, Hazard Class and Packing Group (when applicable). [21] Additionally, the shipping papers must contain an emergency response telephone number and emergency response information to be used in the event of an incident involving hazardous materials in order to assist emergency responders at the scene. The emergency response information must include a basic description (including

technical name, if applicable), immediate hazards to health, risks of fire or explosion, immediate precautions to be taken in the event of an accident or incident, immediate methods for handling fires, initial methods for handling spills or leaks in the absence of fire, and preliminary first aid measures.

Department of Transportation

In addition to OSHA regulations, Calzona/Coastal was required to comply with Department of Transportation (DOT) regulations. The DOT provided regulations that govern tanker operations hauling hazardous materials. [21] More specifically, for Mr. Webb to drive tanker trucks hauling flammable and combustible liquids such as gasoline and diesel fuel, he must have been tested on his knowledge and skills for the commercial motor vehicle license for Group A. This would include passing the written DOT exam; passing the driver's road test; and knowing how to safely load and properly block, brace, and secure the cargo.

As previously stated, in order to haul hazardous materials. Mr. Webb must obtain a state-issued endorsement for tank vehicles which requires a knowledge test followed by obtaining a hazardous materials endorsement. To do so, he must have knowledge of information contained in 49 CFR Parts 171, 172, 173, 177, 178, and 397 on including:

- Hazardous materials regulations;
- Hazardous materials handling;
- Operation of emergency equipment; and,
- Emergency response procedures.

More specifically, 49 CFR Part 177 addresses transportation on a public highway and Part 177.800 addresses the responsibility for compliance and training. This section stated that each carrier/employer shall comply with all the applicable requirements in this part and shall ensure its hazmat employees receive training in relation thereto. It also stated that a carrier/employer may not transport a hazardous material by motor vehicle unless each of its hazmat employees involved in that transportation is trained as required by this part and subpart H of part 172. Subpart H of Part 172 requires the hazmat employee to be trained in the following areas:

- To recognize and identify hazardous materials consistent with the hazard communication standards;
- Function-specific training applicable to the functions the employee performs;
- Emergency response information;
- Measures to protect himself from the hazards associated with the hazardous materials he may be exposed to in the workplace including specific measures the employer has implemented to protect employees against exposure;
- Methods and procedures to avoid accidents;
- Security awareness training; and

- Training conducted by employers as required for compliance with the hazard communication standards required by OSHA (29 CFR 1910.120 or 1910.1200). Training should be provided initially, when job function changes, and every three years thereafter. Each hazmat employer is responsible for compliance with these requirements.

Benzene Standard

The purpose of the OSHA Benzene Standard established in 1987, was to establish uniform requirements for worker health and safety as it applies to the use of benzene containing products which are defined as products containing greater than 0.1 percent. [116] The Benzene Standard specifically excluded fuel distribution terminals that had vapor recovery systems. OSHA recognized that potential benzene exposures at fuel distribution terminals that had vapor recovery systems were substantially below the OSHA benzene action level of 0.5 ppm. [20] "*Thus OSHA concluded that the use of either type of vapor control system would result in average exposures virtually always below the action level and proposed to exempt from this action loading operations at both bulk plants and terminals which use the vapor control systems on this basis.*"

OSHA continued by stating:

> "*OSHA has a high degree of confidence in vapor control technology and has granted the exemption because vapor control technology will keep exposures low. In addition, since very low levels of benzene are ubiquitous, OSHA believes the standard will be more effective if both the standard and the employers' compliance activities are concentrated in areas where there is a likely possibility of exposures over the action level.*"

Prior to the formation of OSHA and promulgation of its initial regulations in 1971, the American Conference of Governmental Industrial Hygienists (ACGIH) had established a Threshold Limit Value (TLV) for benzene. Between 1963 and 1976 the benzene TLV was changed from a 25 ppm 8-hour TWA TLV to a 25 ppm ceiling TLV. Since 1976 the TLV has been reduced to 10 ppm as an 8-hour TWA (1977-1996) and to 0.5 ppm (1997 to the present). [12] The current benzene TLV is recommended to minimize the potential for leukemogenesis (ACGIH 2001). [12]

Table 3 contains a summary of the OSHA PEL and ACGIH TLV for benzene. [117] [12]

Table 3: Benzene Occupational Health Standards and Guidelines

Year	OSHA PEL		ACGIH Benzene TLV	
	8-Hour TWA	STEL/Ceiling	8-Hour TWA	STEL/Ceiling
1971-1976	10 ppm	25 ppm ceiling and 50 ppm 10 min. peak	-	25 ppm

Year	OSHA PEL		ACGIH Benzene TLV	
	8-Hour TWA	STEL/Ceiling	8-Hour TWA	STEL/Ceiling
1977-1980	10 ppm	25 ppm ceiling and 50 ppm 10 min. peak	10 ppm	-
1980-1987	10 ppm	25 ppm ceiling and 50 ppm 10 min. peak	10 ppm	25 ppm
1987-1997	1 ppm	5 ppm STEL	10 ppm	None
1997-Present	1 ppm	5 ppm STEL	0.5 ppm	2.5 ppm STEL

As summarized later in this report, the available literature supported OSHA's opinion regarding potential benzene exposures associated with loading fuels at fuel distribution terminals with vapor recovery systems.

Personal Protective Equipment Standard

The OSHA personal protective equipment standard (29 CFR 1910.132) stated that "*protective equipment, including personal protective equipment for eyes, face, head, and extremities, protective clothing, respiratory devices, and protective shields and barriers, shall be provided, used, and maintained in a sanitary and reliable condition wherever it is necessary by reason of hazards of processes or environment, chemical hazards, radiological hazards, or mechanical irritants encountered in a manner capable of causing injury or impairment in the function of any part of the body through absorption, inhalation or physical contact.*" [118]

According to the OSHA personal protective equipment standard "*the employer shall assess the workplace to determine if hazards are present, or are likely to be present, which necessitate the use of personal protective equipment (PPE).*" [118] If identified hazards are present, then the employer is required to have the affected employees wear the appropriate PPE, communicate selection decisions to each affected employee and train the employees as to what PPE is required, when it is required, how to properly wear the PPE, the limitations of the PPE, and the proper care, maintenance, useful life, and disposal of PPE.

With regards to hand protection, OSHA states (29 CFR 1910.138) "*employers shall select and require employees to use appropriate hand protection when employees' hands are exposed to hazards such as those from skin absorption of harmful substances; severe cuts or lacerations; severe abrasions; punctures; chemical burns; thermal burns; and harmful temperature extremes.*" [119] When selecting hand protection, employers shall base the selection on an evaluation of the performance characteristics of the hand protection relative to the task(s) to be performed, conditions present, duration of use, and the hazards and potential hazards identified.

Exposure Assessment Methodology

The classic definition of industrial hygiene is the science and art related to the anticipation, recognition, evaluation, and control of hazards arising in or from the workplace. Comprehensive

exposure assessment, which is a part of the industrial hygiene process, is the systematic review of the processes, practices, materials, and division of labor present in a workplace that is used to define and judge exposures. [120] In other words, an exposure assessment describes the magnitude (concentration), frequency, and duration of a person's exposure and involves the integration of the work process and environment, the work tasks, the personal protective equipment, and the chemical or physical agents.

As a certified industrial hygienist, I rely upon the following basic tools in order to conduct a generally accepted exposure assessment of personal occupational exposures to persons such as Mr. Webb:

1. a characterization of the environment in which the exposure occurred (including room size and ventilation rate);
2. a characterization of the job and tasks conducted in that environment (including frequency and duration of exposures);
3. a characterization of the products (including volatility);
4. a review and analysis of historical exposure data collected during tasks involving the appropriate handling of the product;
5. evaluation of exposure data to determine whether accepted air sampling and analytical techniques and/or modeling were used to assess the magnitude of exposures; and,
6. a characterization of the relevant safety and health regulations and the associated exposure limits.

The industrial hygienist's training, skills, and experience qualifies him or her to direct efforts for collecting critical information for basic characterization, designating similar exposure groups, and identifying important occupational exposures.

Because it is difficult to measure exposures to every worker, the strategy employed by industrial hygienists is to assemble workers believed to have similar exposures into a Similar Exposure Group (SEG). A SEG is a group of workers having the same general exposure characteristics because of similarities in frequency of the tasks they perform, the materials, and processes with which they work, and the similarity of the way in which they perform the tasks. This process is further defined in the standard industrial hygiene text, *A Strategy for Assessing and Managing Occupational Exposures* and *The Occupational Environment: Its Evaluation and Control*. [121]

Inhalation Exposure Assessment

Several factors must be considered when evaluating the potential occupational exposure to benzene from a specific product or products. These factors include the mass or volume of product used, the percent of benzene contained in the product, the volatility of the material and

the way the product is handled, e.g., spray-applied versus dipped, or poured, and the environment in which it is used.

Air monitoring for determination of employee exposure is presently the only method accepted by OSHA for evaluating compliance with occupational health standards. [120] [122] No air monitoring was ever provided by Mr. Webb's employers and there is no evidence that any air monitoring was ever conducted on him. Therefore, in order to assess Mr. Webb's cumulative benzene exposure, I have had to rely upon the available testimony, discovery documents, and unpublished data and data available in the published literature. There have been several papers published over the past 30 years that have examined fuel transport driver exposures to total hydrocarbon and benzene. [123] [124] [125] [126] [127] It is this literature that is relevant in assessing Mr. Webb's potential exposures. Table 4 contains a summary of the task data from these studies related to locations where there was vapor recovery at the terminal.

Irving and Grumbles (1979) performed a study at 20 US gasoline bulk marketing terminals in 1977 to determine benzene exposures for facility and outside carrier personnel. [124] As part of this study, samples were collected at fueling racks that utilized various methods of product transfer including top loading without vapor recovery, bottom loading without vapor recovery, and bottom loading with vapor recovery. The authors commented that *"[e]mpahsis was placed on taking samples at the rack during loading operations because this is the single procedure which offered the highest potential for exposures."* The benzene content of gasoline from the locations surveyed ranged from 0.5% to 2.4%. The study found that the mean benzene exposure concentration during the bottom loading with vapor recovery was 0.57 ppm. Average loading time was from 15 minutes during bottom loading with vapor recovery. Average benzene concentrations detected during the unloading process was 0.35 ppm without regards to the type of facility. The authors calculated 8-hour TWA benzene concentrations assuming three or four loads. The mean 8-hour time-weighted average (TWA) benzene concentrations was 0.05 ppm (three loads using bottom loading with vapor recovery).

Halder, et al. (1986) collected monitoring data from five US gasoline distribution terminals. [123] Full shift personal sampling was conducted on terminal operators and truck drivers over a twelve-month period and represented various loading systems including bottom loading with vapor recovery, bottom loading with no vapor recovery, and top loading with no vapor recovery. From the 183 collected samples collected from four terminals, the arithmetic mean 8-hour TWA benzene concentration ranged from 0.02 ppm to 0.3 ppm and the geometric mean ranged from 0.02 ppm to 0.3 ppm. The authors found no significant difference in employee benzene exposures between the types of loading systems.

Rappaport, et al. (1987) reported the results of 154 measurements of 55 gasoline components and total hydrocarbons in three job categories: transport driver (n=49), gas station attendants (n=49), and outside operators (n=56) that were collected in 1984. [125] The mean benzene 8-hour TWA air concentration for the transport drivers was 0.14 ppm. The authors reported based on the total

hydrocarbon results for transport drivers that vapor recovery delivery systems had no significant influence on exposures.

Verma, et al. (1992) reported on gasoline and gasoline component exposures that were measured at bulk terminals and agencies at six Ontario petroleum companies in 1986. [127] Bulk terminal and agency drivers are tasked with loading gasoline at terminal and distributing that product to gas stations and other commercial accounts. The benzene content of gasoline ranged from 1% to 3.5%. A total of 59 long term samples were collected (38 terminal drivers and 21 agency drivers) and the arithmetic mean benzene concentration was 0.16 ppm and 0.22 ppm, respectively. The authors did not indicate the distribution of types of loading systems represented by these data except to say that top loading concentrations were higher than bottom loading concentrations.

Verma, et al. (2004) evaluated total hydrocarbon and benzene exposures for petroleum tanker drivers. [126] Exposures were evaluated between 1996 and 1998 for bulk terminal transport drivers in Canada, who transport fuels from primary distribution centers to service stations, commercial accounts and secondary distribution centers, and for agency transport drivers, who transport fuel by small tankers to service stations, smaller commercial accounts, farms, and homes. The average benzene content of gasoline reported by Verma, et al. (2004) during this time period was 1.4% to 1.6%. At the agency, only top loading was used while at most bulk terminals, bottom loading was used while some terminals had vapor control. The authors collected 25 task-based personal samples during bottom loading with vapor recovery and the arithmetic mean and geometric mean benzene concentration were 0.09 ppm and 0.06 ppm, respectively. Additionally, 17 task-based personal samples were collected during the unloading process with vapor recovery and the arithmetic mean and geometric mean benzene concentration were 0.15 ppm and 0.1 ppm, respectively. The authors collected 25 TWA samples with vapor controls. The arithmetic mean and geometric mean TWA benzene concentration was 0.05 ppm and 0.03 ppm, respectively.

As previously stated, air sampling was conducted on 11 and 12 July 2019 during the fuel loading process at the Kinder Morgan/SFPP, Phoenix Terminal. [75] A total of 15 air samples were collected and analyzed in accordance with NIOSH Method 1501 by an AIHA accredited laboratory. Two personal air samples were collected on an operator that shadowed the driver at each loading rack from the time that the driver got out of the truck, during the loading process, and until he got back in the truck to leave the facility. Sampling time ranged from 14 to 23 minutes and all benzene concentrations were below the limit of detection that ranged from less than (<) 0.133 ppm to less than (<) 0.224 ppm.

Table 4: Summary of Bulk Fuel Transport Driver Personal Task Exposure Data

Terminal Type	Source	% Benzene in Gasoline	No. of Samples	Task-Based Benzene Concentration (ppm)		
				Arith. Mean	Geo. Mean	Range
Bottom loading (vapor control)	1	0.5-2.4	-	0.57	-	-
Terminal Driver bottom loading (vapor control)	2	1.4-1.6	25	0.09	0.06	<0.01-0.28
Terminal Bottom Loading (vapor control)	3	-	15	0.085	-	<0.224-<0.13

Source:
1. Irving and Grumbles (1979) [124]
2. Verma, et al. (2004) [126]
3. Kinder Morgan/SFPP Sampling Data [75]

Mr. Webb worked as a tank truck driver for Calzona/Coastal, loading fuel and other products and other hazardous materials between 1 March 1985 and 2 July 2015 or approximately 30.3 years. There was no data or information to indicate the frequency with which Mr. Webb loaded at a specific fuel terminal or the frequency with which he loaded a specific fuel product including gasoline, diesel fuel, jet fuel or avgas. However, based on the following information, I have estimated that 75% of his work involved hauling fuel products and that 50% to 75% of the fuel products that he hauled were gasoline:

- According to Mr. Super, he and Mr. Webb hauled the same products which included chlorine, hydrochloric acid, liquid fertilizer, alcohol, and petroleum products including diesel fuel, different grades of gasoline, jet fuel, and avgas (Deposition of Robert Super, pages 16-17; 26; 32). Mr. Super commented that there were a lot of drivers that only wanted to deliver gasoline or diesel (Deposition of Robert Super, page 37). He and Mr. Webb were two drivers that were willing to deliver fuel products and chemicals (Deposition of Robert Super, page 37).

- When Mr. Webb worked for Calzona and Coastal, he transported more fluids outside the metro areas as compared to his coworkers (Deposition of Mary Major, page 26; Deposition of Jimmy Melvin, page 102).

- According to Mr. Super, Mr. Webb hauled jet fuel on almost a daily basis between 1985 and the early 1990s (Deposition of Robert Super, page 35). During that time all jet fuel for Sky Harbor Airport had to be delivered by truck because there was no pipeline at that time (Deposition of Robert Super, page 33). Sometime in the early 1990s a pipeline was built between the Phoenix Terminal and Sky Harbor Airport (Deposition of Robert Super, page 34). Mr. Super stated that if everything went right, they could deliver three loads of jet fuel during their shift (Deposition of Robert Super, page 34). Because of the low benzene concentration in jet fuel, less than 0.02%, Mr. Webb's benzene exposure would have been well below occupational health standards when loading this fuel product [14]

- Mr. Super testified that there was no avgas in the Phoenix area, but that sometimes drivers who brought the avgas from the West Coast were out of hours. Calzona/Coastal drivers, including Mr. Super and Mr. Webb delivered this fuel to Sky Harbor Airport and smaller airports (Deposition of Robert Super, pages 35-36). Consequently, Mr. Webb would not have loaded avgas.

The total number of years that Mr. Webb hauled gasoline was calculated by multiplying the total number of years he worked (30.3 years) by the percentage of time he hauled fuel products (75%) by the percentage of time he hauled only gasoline (50% to 75%). Consequently, during Mr. Webb's career he hauled gasoline from approximately 11.4 years to 17 years.

There is also uncertainty in the number of times Mr. Webb loaded his truck during any given day. Part of that uncertainty is based on whether Mr. Webb delivered locally or outside the metropolitan Phoenix area. However, based on the following information, I have estimated that Mr. Webb loaded three to six times per day:

- Mr. Super estimated that approximately 25% to 30% of Mr. Webb's trips were outside the metropolitan area (Deposition of Robert Super, page 25). During the last five years that they worked together (1998-2002), Mr. Webb was a dedicated driver for Love's Truck Stops, delivering diesel fuel and gasoline (Deposition of Robert Super, page 29). Most of those truck stops were outside the metropolitan Phoenix area (Deposition of Robert Super, page 91).

- Mr. Super stated that if everything went right, they could deliver three loads of jet fuel during their shift (Deposition of Robert Super, page 34).

- When Mr. Millican worked for Woodland and spent 75% of his time delivering locally and 25% of his time delivering outside Phoenix, he estimated that he loaded five to six loads per day (Deposition of John Millican, page 20).

- Coastal's business model was that the drivers made local deliveries so that they could be home at night (Deposition of Barry Detlefsen, page 24). On a system wide average, drivers haul five to eight loads per day (Deposition of Barry Detlefsen, page 25).

- There were times when a truck would have to wait two to three hours to load (Deposition of Robert Super, page 131). The loading time varied from terminal to terminal.

Mr. Super estimated that the average time it took to load at the terminal was approximately 30 minutes (Deposition of Robert Super, page 211). Furthermore, Mr. Detlefsen stated that drivers would spend less than 45 minutes at a rack, providing they were not waiting in line or if there was no traffic congestion (Deposition of Barry Detlefsen, page 25). Mr. Millican estimated that

it took approximately 15 minutes to load a truck (Deposition of John Millican, page 21). Consequently, I have estimated that Mr. Webb spent 30 minutes loading fuel.

The total time loading fuel per day was calculated by the number of loads (3 to 6) multiplied by the loading time (30 minutes). Consequently, Mr. Webb's fuel loading time ranged from 1.5 hours to three hours per day.

There is some conflicting testimony regarding whether or not Mr. Webb ever top loaded gasoline. Mr. Super, who began working as a tank truck driver in 1979, testified that he and Mr. Webb would have top loaded at the various terminals between 1985 and 1990 (Deposition of Robert Super, pages 173-174). However, according to Mr. Super, he never saw Mr. Webb top loading gasoline at any facility (Deposition of Robert Super, page 258). Other drivers, including Mr. Millican testified that when he loaded at the Phoenix terminal in 1983 there was vapor recovery present but noted that Kinder Morgan/SFPP may have had one or two top loading racks for jet or diesel fuel (Deposition of John Millican, page 25). Also, Mr. Melvin stated that between 1984 and 1985 the only loading rack that still was top loaded was the jet fuel rack and the rest were bottom loading with vapor recovery (Deposition of Jimmy Melvin, page 38). Later, Mr. Melvin testified that all the gasoline racks had been converted to bottom loading racks by approximately 1981 or 1982 (Deposition of Jimmy Melvin, page 112).

The testimony of Mr. Millican and Mr. Melvin is supported by vapor recovery manufacturing specification, drawings, and photographs from 1979 and 1980 which documented that a vapor recovery system was designed and constructed between 1979 and 1980. [61] [62] Consequently, the available information suggested that during Mr. Webb's employment by Calzona/Coastal, he only bottom loaded gasoline at facilities with vapor recovery. Therefore, the only task or shift exposure data relevant to Mr. Webb's potential benzene exposure during loading activities were the data collected at facilities that had bottom loading with vapor recovery. The data specifically relevant to this task includes Irving and Grumbles (1979), Verma, et al. (2004), Kinder Morgan/SFPP (2019). The average of the arithmetic means of these data from facilities that had bottom loading with vapor recovery was 0.25 ppm. Using this data and the time Mr. Webb loaded gasoline, his 8-hour TWA benzene concentration can be calculated as follows:

(loading time x loading concentration) / 8 hours

= (1.5- or 3-hours x 0.25 ppm) / 8-hours

Therefore, his 8-hour TWA benzene exposure would range from 0.047 ppm to 0.094 ppm

Mr. Webb's cumulative benzene exposure was calculated by multiplying his 8-hour TWA exposure concentration (0.047 ppm to 0.094 ppm) by the number of years he loaded gasoline (11.4 years to 17 years). Consequently, Mr. Webb's cumulative benzene exposure when he loaded gasoline ranged from 0.54 ppm-years to 1.6 ppm-years.

I have also calculated Mr. Webb's cumulative benzene exposure for the 15-year period prior to his MDS diagnosis using the same methodology. In this case I multiplied his 8-hour TWA exposure concentration (0.047 ppm to 0.094 ppm) by the number of years he loaded gasoline of the last 15 years (5.63 years to 8.44 years). Consequently, Mr. Webb's cumulative benzene exposure when he loaded gasoline for the 15-year period prior to his MDS diagnosis ranged from 0.26 ppm-years to 0.79 ppm-years.

Report of Plaintiff's Expert Rachael Jones, PhD, CIH

I have reviewed the report of Rachael Jones submitted on behalf of the plaintiff in this case. [128] Dr. Jones provided a review of the records related to this matter including the scientific literature. Based on her review, Dr. Jones opinions can be broken down into two areas:

1. Mr. Webb was exposed to benzene during loading activities and his most likely cumulative exposure ranged from 3.03 ppm-years to 4.7 ppm-years; and,

2. Mr. Webb was not warned of the hazards of benzene, was not informed of his benzene exposure when working at the Phoenix terminal, and, therefore he could not make an informed decision about the risk of developing cancer.

I will address each of these areas.

Mr. Webb was exposed to benzene during loading activities and his most likely cumulative exposure ranged from 3.03 ppm-year to 4.7 ppm-years

Dr. Jones overestimated Mr. Webb's cumulative benzene exposures as summarized below:

1. In her analysis of the literature, Dr. Jones included exposure data from foreign countries. Benzene concentrations in gasoline, particularly European countries, was much higher and on the range of 3% to 4%. [129] [130]

2. Dr. Jones based her cumulative exposure assessment on the assumption that Mr. Webb conducted top loading between 1985 and 1987. However, as previously stated, the testimony of Mr. Millican and Mr. Melvin about facilities having only bottom loading and vapor recovery during Mr. Webb's employment, is supported by vapor recovery manufacturing specification, drawings, and photographs from 1979 and 1980 which document that a vapor recovery system was designed and constructed between 1979 and 1980. [61] [62] Consequently, the best available information indicated that during Mr. Webb's employment by Calzona/Coastal, he only bottom loaded gasoline at facilities with vapor recovery. Therefore, the only task or shift exposure data relevant to Mr. Webb's potential benzene exposure during loading activities are the data collected at facilities that had vapor recovery. Thus, Dr. Jones's

reliance of air sampling data collected during top loading operations has resulted in an overestimation of Mr. Webb's cumulative exposure.

Mr. Webb was not warned of the hazards of benzene, was not informed of his benzene exposure when working at the Phoenix terminal, and, therefore he could not make an informed decision about the risk of developing cancer

Mr. Webb was provided training on the hazards of the fuel products he hauled by his employers, Calzona/Coastal and it was not the responsibility of the terminal owners to provide warnings to him about benzene for several different reasons:

1. As previously stated, this matter involved the transportation and potential exposures to gasoline, diesel fuel, and avgas, not benzene. These fuel products have been tested as a whole and agencies such as NTP, IARC, and ACGIH, who review chemicals for carcinogenicity, have reviewed the literature on these products and concluded that it does not cause any benzene related cancer even though it contained limited amounts of benzene. [14] [15] [12] [16]

2. Under OSHA's Hazard Communication Standard, gasoline is considered to be a mixture because it is a combination of two or more chemicals that is not the result of a chemical reaction. Furthermore, because gasoline and the other fuel products have been tested as a mixture, the hazard communication standard requires manufacturers or importers to use that test data to determine their hazards. Therefore, it was not necessary to include a separate warning for benzene.

3. Material Safety Data Sheets for the fuel products were provided to Mr. Webb's employers, Calzona and Coastal. As sophisticated fuel carriers, it was Mr. Webb's employer's responsibility to train their employee on the hazards of the products that they hauled.

4. OSHA's Benzene Standard specifically excluded fuel distribution terminals that had vapor recovery systems. OSHA recognized that potential benzene exposures at fuel distribution terminals that had vapor recovery systems were substantially below the OSHA benzene action level of 0.5 ppm. [20] "*Thus OSHA concluded that the use of either type of vapor control system would result in average exposures virtually always below the action level and proposed to exempt from this action loading operations at both bulk plants and terminals which use the vapor control systems on this basis.*"

   OSHA did provide an exception in that employers are required to include the provisions of OSHA's Hazard Communication Standard 29 CFR 1910.1200 as incorporated into this section. Therefore, it was the responsibility of Mr. Webb's employers to comply with the information and training requirements.

5. Based on the testimony of Mr. Super, he was aware of the hazards of gasoline and benzene. Mr. Super noted that he had seen signs at loading racks and gasoline stations, which indicated that gasoline contained chemicals that were known to cause cancer (Deposition of Robert Super, page 122). The signs were related to animal studies and kidney cancer, not benzene-related cancers. Mr. Super also knew that gasoline and avgas contained benzene (Deposition of Robert Super, page 122). He stated that it was common knowledge to him and hazmat drivers like Mr. Webb that a chemical like benzene was potentially harmful to their health (Deposition of Robert Super, page 123). Mr. Super stated that it was early in his career that he learned that benzene was a component of gasoline (Deposition of Robert Super, page 210).

Summary of Opinions

Based upon my review of the case-specific documents and the scientific literature I am offering the following opinions to a reasonable degree of scientific certainty:

- This matter involved the transport of gasoline, diesel fuel, jet fuel and avgas, not benzene. Based on standard industrial hygiene references, occupational exposures to these fuel products are not associated with Myelodysplastic Syndrome (MDS).

- Based on the available scientific literature related to tank truck drivers loading gasoline, Mr. Webb's benzene exposure when loading gasoline was less than approximately 0.047 ppm to 0.094 ppm as an 8-hour TWA concentration. This long-term benzene concentration was well below the OSHA 8-hour TWA PEL of 1 ppm and the ACGIH TLV of 0.5 ppm.

- Mr. Webb's reasonable worst-case cumulative benzene exposure to benzene from loading gasoline ranged from 0.54 ppm-years to 1.6 ppm-years. Additionally, Mr. Webb's cumulative benzene exposure when he loaded gasoline for the 15-year period prior to his MDS diagnosis ranged from 0.26 ppm-years to 0.79 ppm-years.

- Under the OSHA Act of 1970, Mr. Webb's employers had the responsibility to provide education and training regarding the safe work practices with the products, evaluate the potential exposures encountered by its employees, and ultimately were responsible for protecting the health of its employees. Additionally, Department of Transportation requires hazardous material transport employees to have training including recognizing and identifying hazardous materials, function-specific training applicable to the functions the employee performs, emergency response procedures, measures to protect himself from the hazards associated with the hazardous materials he may be exposed to in the workplace, methods and procedures to avoid accidents, and security awareness training.

- Plaintiff's industrial hygiene expert, Dr. Rachael Jones, by incorporating benzene exposure data from foreign countries and assuming that Mr. Webb bottom loaded

gasoline without vapor recovery, significantly overestimated Mr. Webb's cumulative exposure.

- Dr. Jones based her cumulative exposure assessment on the assumption that Mr. Webb conducted top loading between 1985 and 1987. However, the weight of the evidence suggested that during Mr. Webb's employment by Calzona/Coastal, he only bottom loaded gasoline at facilities with vapor recovery. Therefore, the only task or shift exposure data relevant to Mr. Webb's potential benzene exposure during loading activities are the data collected at facilities that had vapor recovery. Thus, Dr. Jones's reliance of air sampling data collected during top loading operations has resulted in an overestimation of Mr. Webb's cumulative exposure.

- Dr. Jones opined that Mr. Webb was not warned of the hazards of benzene, was not informed of his benzene exposure when working at the Phoenix terminal, and, therefore he could not make an informed decision about the risk of developing cancer. However, the terminal operators provided Mr. Webb's employers and Mr. Webb with access to gasoline MSDS. As sophisticated fuel carriers, it was Mr. Webb's employer's responsibility to train their employees, including Mr. Webb, on the hazards of the products that they hauled.

My opinions are to a reasonable degree of scientific certainty and are based on my more than 42 years of experience as an industrial hygienist and safety professional. My experience has included health hazard evaluations and audits of multiple operations within facilities similar to those where Mr. Webb was employed. My experience has also included the development of exposure assessment strategies, training of employees who worked in numerous industrial operations, my prior work as a member of the AIHA's Product Safety and Health Committee, and my current membership in the Society for Chemical Hazard Communication. I also base my opinion upon portions of the scientific literature focused on hazard communication and occupational health hazard assessment.

For purposes of this report, I have reviewed numerous documents, articles, studies and publications, which include but are not limited to the following:

[1] Complaint.
[2] Plaintiffs' Responses to Defendant Pro-Petroleum, Inc.'s Uniform and Non-Uniform Interrogatories to Plaintiffs.
[3] Deposition of Mary Major, taken 7 March 2019.
[4] Deposition of Joshua Webb, taken 7 March 2019.
[5] Deposition of Eugene Martin, taken 6 March 2019.
[6] Deposition of Jimmy Melvin, taken 6 March 2019.

[7] Deposition of Robert Super, taken 15 April 2019.

[8] Deposition of Barry Detlefsen, taken 8 January 2019.

[9] Coastal/Calzona Employment Records for Elwyn Webb (Coastal0001-Coastal2915).

[10] Calzona Tankways. Application for Employment (Coastal0463-Coastal0466).

[11] Coastal Transportation Co. Payroll Checklist Worksheet (Coastal0189).

[12] American Conference of Governmental Industrial Hygienists (ACGIH). 2001. Documentation of the Threshold Limit Value and Biological Exposure Indices for Gasoline.

[13] Runion, H.E. 1975., "Benzene in Gasoline," *American Industrial Hygiene Journal,* vol. 37, pp. 338-350, 1975.

[14] International Agency for the Research on Cancer (IARC), Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 45, 1989.

[15] National Toxicology Program (NTP) Report on Carcinogens, Fourteenth Edition, 2016.

[16] Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Gasoline. 1995.

[17] Agency for Toxic Substances and Disease Registry (ATSDR). Toxicoloigcal Profile for Fuel Oils. 1995.

[18] American Conference of Governmental Indsutrial Hygienists (ACGIH). 2003. Documentation of the TLVs and Biological Exposure Indices for Kerosene/Jet Fuel.

[19] American Conference of Governmental Industrial Hygienists (ACGIH). 2019. TLVs and BEIs Based on the Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. ACGIH: Cincinnati, OH.

[20] Federal Register. Vol. 52, No. 176, p. 34460-34578. Department of Labor, Occupational Safety and Health Administration. 29 CFR Part 1910. Occupational Exposure to Benzene; Final Rule. 11 September 1987.

[21] Department of Transportation, Federal Motor Carrier Safety Administration 49 CFR Parts 171, 172, 173, 177, 178, 383, and 397.

[22] Certification of Road and Written Tests for Elwyn Webb (Coastal0477-Coastal0481).

[23] DOT Commercial Drivers License Renewal, Issued 27 December 1991 (Coastal0233).

[24] Coastal Transport Co. Driver Training Manual dated 2014 (Coastal0851-Coastal1263).

[25] Coastal Transportation, Policies and Procedures for Driver Personnel, Revised April 2015 (Coastal1271-Coastal1322).

[26] Calzona First Responsder Awareness Training for Elwyn Webb, (Coastal0268).

[27] Calzona Certificate for Elvyn Webb for Attending a 16-hour Hazardous Waste Transportation Course, dated 10 July 1992 (Coastal0283-Coastal0284).

[28] Coastal Transportation Training Certificate for Elwyn Webb, dated 26 February 2013 (Coastal0493-Coastal0499).

[29] Coastal Transportation Training Certificate for Elwyn Webb, dated 12 January 2010

(Coastal0502-Coastal0509).

[30] Coastal Transportation Training Certificate for Elwyn Webb, dated 17 January 2007 (Coastal0511-Coastal0518).

[31] Coastal Transportation Training Certificate for Elwyn Webb, dated 28 January 2004 (Coastal0519-Coastal0532).

[32] Coastal Transportation Training Certificate for Elwyn Webb, dated 26 February 2001 (Coastal0534-Coastal0550).

[33] Coastal Transportation Training Certificate for Elwyn Webb, dated 10 August 1998 (Coastal0551-Coastal0556).

[34] Defendant ConocoPhillips Company's Second Supplement Rule 26.1 Disclosure Statement.

[35] Deposition of Douglas Kleopfer, taken 29 May 2019.

[36] Deposition of Dennis Gilmore, taken 31 May 2019.

[37] Tosco Corporation Memo Regarding Gasoline Benzene Limit dated 19 July 1988 (PSXMAJ00007964).

[38] Tosco Benzene Policy, dated February 1995 (PSXMAJ00008000-PSXMAJ00008014).

[39] Tosco Carrier Access and Loading Agreement, dated 8 march 1999 (PSXMAJ00000001-PSXMAJ00000003).

[40] ConocoPhillips Terminal Access Agreement with Coastal, dated 1 December 2005 (PSXMAJ00000035-PSXMAJ00000039).

[41] Tosco Load Rack & Driver Laoding Card Agreement for Elwym Webb, dated 13 June 1997 (PSXMAJ00005044).

[42] Tosco Load Rack & Driver Loading Card Agreement for Elwyn Webb, dated 10 Julyu 2001 (PSXMAJ00005043).

[43] Tosco Tank Truck Loading Pocedures, dated 17 January 2001 (PSMAJ00001577-PSMAJ00001581).

[44] Tosco Truck Loading Rack Operations, dated 31 July 2008 (PSXMAJ00001521-PSXMAJ00001528).

[45] Maricopa County Air Quality Department Inspection dated 20 December 2005 (PSXMAJ00000751-PSXMAJ00000753).

[46] ConocoPhillips Daily Operator Tasks (PSXMAJ00004594-PSXMAJ00004610).

[47] ConocoPhillips Weekly Facility Inspection Reports (PSXMAJ00004661-PSXMAJ00004662; PSXMAJ00004668; PSXMAJ00006384-PSXMAJ00006405; PSXMAJ00006334-PSXMAJ00006411).

[48] ConocoPhillips Monthly Load Rack Inspection Report (PSXMAJ00000869-PSXMAJ00000929; PSXMAJ00004471; PSXMAJ00002358-PSXMAJ00002358-PSXMAJ00002549; PSXMAJ00004383-PSXMAJ00004431; PSXMAJ00004471-PSXMAJ00004493).

[49] Tosco Monthly Monitoring (PSXMAJ00004471-PSXMAJ00004493).

[50] Tosco Memo Regarding Benzene Analysis, dated 26 May 1977 (PSXMAJ00007965-PSXMAJ00007966).

[51] Tosco Corporation. Industrial Hygiene Management Plan, dated 1 June 2001 (PSXMAJ00007967-PSXMAJ00007999).

[52] Tosco Sampling Data Spreadsheet (PSXMAJ00000004-PSXMAJ00000006).

[53] Phillips 66 Air Sampling Memo, date 30 January 2002 (PSXMAJ00004982-PSXMAJ00004985).

[54] Tosco Employee Notification of Monitoring Results memo (PSXMAJ00004986-PSXMAJ00004994).

[55] ConocoPhillips personal IH Monitoring Memo, dated 3 January 2006 (PSXMAJ00004981).

[56] Phillips 66 Gasoline Material Safety Data Sheet, dated 26 February 1999 (PSXMAJ00003235-PSXMAJ00003242).

[57] Phillips 66 Gasoline Material Safety Data Sheet, dated 11 May 2001 (PSXMAJ00003213-PSXMAJ00003220).

[58] Unbranded Gasoline Material Safety Data Sheet, dated 26 September 2002 (PSXMAJ00003271-PSXMAJ00003281).

[59] Unbranded Gasoline Material Safety Data Sheet, dated 1 January 2003 (PSXMAJ00004364-PSXMAJ00004374).

[60] K. M. B. o. L. (KinderMorgan005678-KinderMorgan006003).

[61] HydroTech Engineering. Manufacturing Specifications for Packaged Vapor Recovery System, dated 22 August 1979.

[62] Kinder Morgan Vapor Recovery Plans and Photographs, dated 1980.

[63] Deposition of Casey Alleman, taken on 15 November 2015.

[64] Kinder Morgan Driver Safety Class Rosters dated 15 April 2009 and 18 July 2012 (KinderMorgan000159-KinderMorgan000161; KinderMorgan000127).

[65] Defendant SFPP, LP and Kinder Morgan's Sixth Supplemental Disclosure Statement.

[66] Kinder Morgan Loading and Unloading Safety Rules and Procedures, Revised 3 March 2016 (KinderMorgan000001-KinderMorgan000015).

[67] Kinder Morgan Product Truck Loading Procedures and Acknowledgment, revised 9 November 2016 (KinderMorgan006263-KinderMorgan006268).

[68] Kinder Morgan Petroleum Substances Handling, revised 13 September 2017 (KinderMorgan006312-KinderMorgan006319).

[69] Kinder Morgan Hazard Communication, revised 13 January 2016 (KinderMorgan006320-KinderMorgan006326).

[70] Kinder Morgan Phoenix Terminal Daily Operations Checklist (KinderMoran006004-KinderMorgan006262; KinderMorgan007718-KinderMorgan008021).

[71] Kinder Morgan Phoenix Terminal Loading Rack/Vapor Recovery Equipment Repair Sheet (KinderMorgan006376-KinderMorgan006383; KinderMorgan0076850KinderMorgan007717).

[72] Axial Technical Report for Santa Fe Pacific Corporation, dated 5 November 1991 (KinderMorgan016446-KinderMorgan016465).

[73] URS Greiner Woodward Clyde Industrial Hygiene Survey Report for Kinder Morgan Terminal, dated 8 July 1999 (KinderMorgan004182-KinderMorgan004192).

[74] Corporate Shared Services EHS Industrial Hygiene Report for Kinder Morgan Phoenix Terminal, conducted June 2015 (KinderMorgan000049-KinderMorgan000123).

[75] CTEH Air Sampling Data from the Kinder Morgan Phoenix Terminal, conducted on 11 and 12 July 2019.

[76] Caljet's Fifth Supplemental Disclosure Statement.

[77] Deposition of John Millican, taken on 8 February 2019.

[78] Caljet Rack Loading Procedures (CJ002990-CJ002991).

[79] Deposition of Michael Gray, taken on 8 February 2019.

[80] Caljet Inspection and Testing Reports (CJ002406-CJ005207; CJ005290-CJ005762; CJ005845--CJ006023).

[81] Assay Technology Laboratory Report dated 9 April 2012 (CJ001839).

[82] ATC Benzene Exposure Assessment at the Cal-Jet Terminal Phoenix Tank Farm, dated 15 July 2019.

[83] Defendant Pro Petroleum, Inc's Third Supplemental Rule 26.1 Disclosure Statement.

[84] Defendant Pro Petroleum's Responses to Plaintiff's First Uniform Interrogatories, Non-Uniform Interragatories & Request for Production.

[85] Pro Petroleum, Inc. General Safety Rules Truck Loading/Unloading Operating Instructions Driver Carding Requirements, updated 30 May 2013 (Major/ProPetrol00035-Major/ProPetrol00053).

[86] Pro Petroleum, Inc. General Safety Rules Truck Loading/Unloading Operating Instructions Driver Carding Requirements, Updated 25 June 2018 (Major/ProPetrol00054-Major/ProPetrol00072).

[87] Pro Petroleum, Inc. General Safety Rules Truck Loading/Unloading Operating Instructions Driver Carding Requirements, Updated 24 July 2018 (Major/ProPetrol00073-Major/ProPetrol00091).

[88] Pro Petroleum Quarterly Calibration Sign Off Sheet (Major/ProPetro00029).

[89] Pro Petroleum Vapor Combustion Unit daily Operation Log (Major/ProPetrol00030).

[90] Pro Petroleum Weekly Gas Detection Sign Off Sheet (Major/ProPetrol00031).

[91] Pro Petroleum Weekly Leak Detection and Repair Program Log (Major/ProPetrol00032).

[92] Pro Petroleum Weekly Load Rack Inspection (Major/ProPetrol00034).

[93] ATC Benzene Exposure Assessment at the Pro Petroleum Terminal, Issued 19 July 2019

(Major/ProPetrol00097-Major/ProPetrol00113).

[94] Chevron Material Safety Data Sheet for Diesel Fuel, Revision Date 7 May 2015 (Major/ProPetrol00018-Major/ProPetrol00028).

[95] Marathon Petroleum Ultra Low Sulfur Diesel with Biodiesel, Revision Date 27 May 2016 (Major/ProPetrol00001-Major/ProPetrol00017).

[96] Marathon Material Safety Data Sheet for Petroleum Aviation Turbine Jet Fuel, dated 1 June 2016.

[97] Defendant Circle K Terminal LLC's Initial Rule 26.1 Disclosure Statement.

[98] Deposition of Scott Mitchell, taken 28 November 2018.

[99] Circle K Termainal Truck Loading Procedure Overview. Revision Date 1 June 2010 (Exhibit 6 to Mitchell Depositions).

[100] ATC. Benzene Exposure Assessment at the Circle K Terminal at Phoenix Tank Farm, dated 9 July 2019.

[101] Valeros Marketing and Supply Company's Rule 26.1 Initial Disclosure Statement.

[102] Transportation Service Agreement between Valero and Coastal Transportation, (Valero000001-Valero000029).

[103] Deposition of Melinda Farias, taken 19 June 2019.

[104] Deposition of Matthew Hodges, taken on 19 June 2019.

[105] Valero Benzene in Gasoline Data (Valero000645-Valero000792).

[106] Diamond Shamrock Material Safety Data Sheet for Gasoline dated 1 January 2002 (Valero000477-Valero000485).

[107] Valero Material Safety Data Sheet for Unleaded CARB Gasoline, dated 28 November 2005 (Valero000581-Valero000595).

[108] Valero Material Safety Data Sheet for Unleaded Gasoline, dated 28 November 2005 (Valero000630-Valero000644).

[109] Valero Material Safety Data Sheet for Unleaded Gasoline, dated 9 February 2009 (Valero000613-Valero000629).

[110] Valero Material Safety Data Sheet for Unleaded Gasoline, dated 5 January 2010 (Valero000596-Valero000612).

[111] Valero Unleaded Gasoline Material Safety Data Sheet, dated 28 July 2011 (Valero000313-Valero000329).

[112] Valero Material Safety Data Sheet for Unleaded Gasoline, dated 23 May 2014 (Valero000507-Valero000521).

[113] Henshaw, J.L., Gaffney, S.H., Madl, A.K., Paustenbach, D.J., "The Employer's Responsibility to Maintain a Safe and Healthful Work Environment: An Historical Review of Societal Expectations and Industrial Practices." Employ Response Rights J (2007).

[114] The Occupational Safety and Health Act, 1970.

[115] OSHA 29 Code of Federal Regulations (CFR) 1910.1200

[116] OSHA 29 Code of Federal Regulations (CFR) 1910.1028.

[117] OSHA 29 Code of Federal Regulations (CFR) 1910.1000.

[118] OSHA 29 Code of Federal Regulations (CFR) 1910.132.

[119] OSHA 29 Code of Federal Regulations (CFR) 1910.138.

[120] S. DiNardi, Ed., The Occupational Environment - Its Evaluation and Control, Fairfax, VA, 1997.

[121] S. D. Jahn, W. H. Bullock and J. S. Ignacio, Eds., A Strategy for Assessing and Managing Occupational Exposures, Fourth Edition, Falls Church, VA: American Industrial Hygiene Association, 2015.

[122] OSHA Technical Manual, Section II: Chapter 1, Introduction.

[123] Halder, C.A., Van Gorp, G.S., Hatoum, N.S., Warne, T.M., "Gasoline Vapor Exposures. Part I. Characterization of Workplace Exposures," *American Industrial Hygiene Association Journal,* vol. 47, pp. 164-172, 1986.

[124] Irving, W.S. and Grumbles, T.G., "Bulk Exposures During Gasoline Loading at Bulk Marketing Terminals," *American Industrial Hygiene Association Journal,* vol. 40, pp. 468-473, 1979.

[125] Rappaport, S.M., Selvin, S., Waaters, M.A., "Exposures to Hydrocarbon Components of Gasoline in the Petroleum Industry," *Applied Industrial Hygiene*, vol. 2, pp. 148-154, 1987.

[126] Verma, D.K., Cheng, W.K., Shaw, D.S., Shaw, M.L., Verma, P., Julian, J.A., Dumschat, R.E., Mulligan, S.J.P., "A Simultaneous Job- and Task-Based Exposure Evaluation of Petroleum Tanker Drivers to Benzene and Total Hydrocarbons," *Journal of Occupational and Environmental Hygiene*, vol. 1, pp. 725-737, 2004.

[127] Verma, D.K., Julian, J.A., Bebee, G., Cheng, W.K., Holborn, K., Shaw, L., "Hydrocarbon Exposures at Petroleum Bulk Terminals and Agencies," *American Industrial Hygiene Association Journal,* vol. 53, pp. 645-656, 1992.

[128] Expert Report of Rachael Jones, PhD, CIH, dated 21 May 2019.

[129] Hakkola, M. and Saarinen, L. 1996. "Exposure of Tanker Drivers to Gasoline and Some of its Components." Annals of Occupational Hygiene. 40:1-10.

[130] Nordlinder, R. and Ramnas, O. 1987. Exposure to Benzene at Different Work Places in Sweden. Journal of Occupational Hygiene. 31:345-355.

[131] Various occupational safety and health publication and articles developed by governmental agencies, professional and trade associations, voluntary consensus standards organizations, and researchers.

This report is based on the information available to me at this time. Should additional information become available, I reserve the right to determine the impact, if any, of the new information on my opinions and conclusions, including the opinion and conclusions regarding Mr. Webb's occupational exposures, and to revise my opinions and conclusions if necessary.

Sincerely,

John W. Spencer, CIH, CSP
President

ATTACHMENT 1

Curriculum Vitae

EPI

8805 Columbia 100 Pkwy
Suite 100
Columbia, MD 21045

(410) 740-9600
FAX (410)740-9606

www.episervices.com

CURRICULUM VITAE

JOHN W. SPENCER, CIH, CSP

Date of Birth: 12 February 1954
Citizenship: USA

Education:

1980-1981	National Institute for Occupational Safety and Health and OSHA Training Institutes – Special Programs
1973-1976	B.S. Biological Sciences University of Maryland College Park, Maryland
1972-1973	St. Mary's College St. Mary's City, Maryland

Professional Experience:

1993 - Present	President Environmental Profiles, Inc. Columbia, Maryland
June 1990 - 1993	Vice President and Director of Environmental Sciences National Medical Advisory Service Bethesda, Maryland
1988-1990	Principal Daft-McCune-Walker, Inc. Towson, Maryland President DMW Environmental Services, Inc. a subsidiary of Daft-McCune-Walker
1987-1988	Corporate Industrial Hygienist and Environmental Coordinator United States Fidelity and Guarantee Company Baltimore, Maryland
1982-1987	Director of Industrial Hygiene and Occupational Health Programs United States Coast Guard, 5th District Portsmouth, Virginia

Professional Experience (cont.):

1980-1982	Team Leader/Industrial Hygienist National Institute for Occupational Safety and Health National Occupational Hazard Survey Cincinnati, Ohio
1977-1980	Industrial Hygienist Equitable Environmental Health Rockville, Maryland

Certifications and Registrations:

1987	American Board of Industrial Hygiene Certified Industrial Hygienist
1991	Board of Certified Safety Professionals Certified Safety Professional

Professional Societies:

	American Indoor Air Quality Council
	American Industrial Hygiene Association
	American Board of Industrial Hygiene
	American Conference of Governmental Industrial Hygienists
	Board of Certified Safety Professionals
	American Association for the Advancement of Science
	Society for Chemical Hazard Communication
1999	Member, American Society of Safety Engineers
1998	Member, American Association for the Advancement of Science
1996	Member, New York Academy of Sciences
1993-94	Member, Maryland Industrial Hygiene Council
1992-93	President, American Industrial Hygiene Association, Chesapeake Section
1992	President-Elect, American Industrial Hygiene Association, Chesapeake Section

Committees:

American Industrial Hygiene Association:
Product Health and Safety Committee (1991-1995)
- MSDS and Labeling and other Warning Issues
Emergency Response Planning Committee (1991-1999)

Awards:

1987	USF&G Company Excellence Through Service Award
1976	National Institutes of Health Outstanding Achievement Award

Selected Project Management Experience:

2001	Director of health, safety, and environmental management for a ship recycling firm. Managed the proper removal of asbestos, PCB, mercury, lead, petroleum products, and other regulated substances.
1997	Planned and conducted facility audits for health and safety regulatory requirements and Voluntary Protection Programs elements. Completed eleven (11) facilities in a three-week period using in-house developed software auditing and tracking tools.
1994-1996	Developed and implemented exposure assessment strategies of film processing operations. The operations included mass color film processing, and color film processing during the operation of a minilab. Investigations have also included the review of potential chemical exposures resulting from the use of X-ray development equipment in private doctors' offices and hospital environments.
1994	Conducted oversight of the environmental clean up of a U.S. naval aircraft carrier during a shipbreaking process. Evaluated for contaminated waters, painted surfaces, PCB, and asbestos containing materials. Insured the proper removal and disposal of all waste materials.
	Developed product warning labels and material safety data sheets for industrial and consumer products.
	Managed the final clearance of asbestos from approximately 25 occupied apartment buildings. Oversaw clean-up strategy, including air monitoring of work and adjacent spaces.

Selected Project Management Experience (cont.):

1994	Have conducted numerous indoor air quality investigations of commercial office space, clinical laboratories, and on University campuses. Assessments included review of the heating ventilating and air condition system, management programs to respond to IAQ complaints and real time monitoring for chemical, physical, and biological agents.
1994	Conducted audits of health, safety, environmental and management programs of multiple chemical processing facilities.
1991, 1992, 1993	Designed and implemented several comprehensive product risk analysis evaluations for product manufacturers. Analysis included hazard identification, toxicological assessments, industrial hygiene exposure assessment, and risk characterization. Recommendations to control or eliminate potential user exposures were provided.
1991, 1992, 1993	Supplemental information for product warnings by the MSDS, labels, and technical information bulletins was also included.
1990, 1991, 1993	Provided expert opinion on sufficiency of labels and warnings for chlorinated solvents, isocyanate, and benzene containing products.
1989	Evaluated a 450-acre manufacturing facility with nearly 3 million square feet of manufacturing and warehouse space for hazardous substances which may have represented liability to the potential purchaser under CERCLA. Reported directly to the Rouse Company in Columbia, Maryland as their environmental advisor for the approximate $43 million property transfer.
1988-1990	Have conducted numerous exposure assessments to evaluate actual personal exposure levels that resulted from various workplace tasks and environments. Benzene, asbestos, formaldehyde, chlorinated solvents, and automobile by-products of combustion were evaluated via real-time assessments to assess actual personal exposures.
1988-1989	Developed a groundwater monitoring and protection program for a new golf course facility. Determined environmental base line parameters to be applied to subsequent future groundwater sampling. Assessed pesticide environmental fate mechanisms and degradates resultant from turf management practices.

Selected Project Management Experience (cont.):

1988-1989	Evaluated hazardous material haulers exposure to cargo during pick-up, transit, and off-loading. Established recommendations for personal protective equipment and work practices to reduce and eliminate significant exposures to cargo. Chemicals evaluated included the isocyanates, MDI and TDI and methylene chloride.
1986	Conducted a detailed health hazard evaluation of an EPA Superfund (CERCLA) site in New Jersey. Monitored hazardous waste site workers' exposure to a multitude of chemical contaminants.
1985-1987	Development and implementation of Occupational Medical Monitoring, Hearing Conservation, Lead, Asbestos and Hazard Communication programs for approximately 4,000 military and civilian personnel involved in manufacturing, office and residential environments. Measured exposures to benzene, aliphatic hydrocarbons, and other chemical and physical agents in industrial and shipboard environments.
1985	Conducted Asbestos and Lead Training Programs for shipyard workers involved in abatement procedures. Instructed workers in the areas of potential health hazards, health and safety measures and methods for reducing their exposure. Prepared labels for in-house product use.
1980-1982	Led a team of seven industrial hygienists in the NIOSH National Occupational Hazard Survey. My team visited approximately 1,500 facilities across the United States. We reviewed management practices related to employee safety and health, conducted wall-to-wall audits of the facility, reviewed product labels and MSDS, inventoried products and their constituents from readily available information and developed a product database.
1979	Conducted a wall-to-wall survey of a pharmaceutical facility evaluating worker exposures and recommended methods for regulatory compliance.

Professional Development Courses:

REACH - A Risk Management Strategy, June 2014, American Industrial Hygiene Conference Exposition.

Risk Assessment Symposium, AIHA 6-7 November 2008, Tampa, FL

Introduction to Monte Carlo Uncertainty Analysis, PDC 8, 26 September 1999, PCIH '99

Risk Assessment, PDC 6, 26 September 1999, PCIH '99

Professional Development Courses (cont.):

Mathematical Models for Occupational Exposure Assessment, PDC 402, 6 June 1999, AIHCE
International Hazard Communication, AIHA/SCHC (12 hrs)

Environmental Toxicology, Hood College (24 hrs)

Man-Made Mineral Fibers: Status of Health Risk Assessment, Johns Hopkins University (12.5 hrs)

Health Hazard Recognition & Evaluation, OSHA Institute (80 hrs)

Health Hazard Recognition & Evaluation, NIOSH Training Institute (80 hrs)

Chemical Process Industries, University of Cincinnati (40 hrs)

Industrial Ventilation Conference, North Carolina State University (40 hrs)

Mechanisms of Toxicology, Johns Hopkins University (25 hrs)

Asbestos Symposium, Johns Hopkins University (8 hrs)

Loss Control Management, U. S. Coast Guard (40 hrs)

Pulmonary Medicine Topics, U.S. Navy Conference (8 hrs)

Navy Occupational & Environmental Health Workshop, U.S. Navy Conference (40 hrs)

Comprehensive Review of Industrial Hygiene, University of Utah (40 hrs)

Air Surveillance for Hazardous Materials, U.S. EPA (40 hrs)

Appropriate IH Data Collection for Future Occupational Epidemiology Studies (4 hrs)

Certified Indoor Air Quality Consultant Study/Review Course (20 hrs)

Radiological Emergency Management, FEMA (10 hrs)

Effective Communication, FEMA (8 hrs)

Introduction to CERT, FEMA (8 hrs)

An Introduction to Hazardous Materials, FEMA (10 hrs)

Selected Speaking Engagements:

2010 Bilenki, A.M. & Spencer, J.W., "Dental Technician Exposure to Beryllium using Dental Casting Alloys." American Industrial Hygiene Conference and Exposition, Denver, Colorado, May 2010.

Selected Speaking Engagements (cont.):

Burrelli, L.G., Driscoll, C.T., & Spencer, J.W., "Asbestos Crane Friction Debris Study." American Industrial Hygiene Conference and Exposition, Denver, Colorado, May 2010.

Plisko, M.J. & Spencer, J.W.,"TWA or STEL? A Measurement Strategy for Determining VOC Exposure during use of a Mixed-hydrocarbon Solvent." American Industrial Hygiene Conference and Exposition, Denver, Colorado, May 2010

2009 "Dermal Modeling for Solvents – The Limitation of Applications for Practicing Industrial Hygienists" American Industrial Hygiene Conference and Exposition, Toronto, Canada. June 2009

Plisko, M.J., Spencer, J.W., & Nealley, M.L. "How do my Predictions Compare? Correlating Predictive Modeling with Actual Results." American Industrial Hygiene Conference and Exposition, Toronto, Canada. June 2009.

2007 "A Validation Study of a Mathematical Model for Estimating Solvent Exposures in the Workplace." American Industrial Hygiene Conference and Exposition, June 2007.

"The Implications of Input Variables Selection When Modeling Occupational Exposures." American Industrial Hygiene Conference and Exposition, June 2007.

2003 "Estimating Past Exposures- The Scientific Basis for Reconstructing Asbestos Dose for Groups and Individuals." American Industrial Hygiene Conference, May 2003

2002 "Where do we start? The proper response to an indoor air quality complaint. Investigation and testing techniques; determining causes; remediation," 18th Annual Maryland Workers' Compensation Educational Association Inc. Conference, 24 September 2002.

2001 "Generating Exposure Data on Historical Activities or Products", American Industrial Hygiene Conference, 4 June 2001.

"Evaluation of Chemical Exposures in Mammography X-Ray Development," American Industrial Hygiene Conference, 4 June 2001.

Selected Speaking Engagements (cont.):

	"Comparison of Direct and Indirect Sample Preparation Methods for Asbestos Analysis", American Industrial Hygiene Conference, 6 June
1999	"The Actual Contribution of Airborne Asbestos Fibers to the Work Environment from Asbestos Gaskets", American Industrial Hygiene Conference and Exposition, 7 June 1999.
1998	Federal Safety and Health Council of Central Maryland Health & Safety Programs: Auditing, Self-Assessments and Issues Tracking
1995	"Environmental Health & Safety Auditing — Performance Measures," Program Chairperson, Johns Hopkins University, Baltimore, Maryland, October 1995
	"Health & Safety Audits Course", Program Chairperson, Government Institute, Orlando, Florida, February 1995
	"Issues Critical to Growth", Maryland Chamber of Commerce, Baltimore Leadership Training, Baltimore, MD, 15 May 1995
1994	"Health and Safety Compliance Auditing Course", 3 days UNOCAL Corporation, Los Angeles, CA, August & September, 1994
	"Indoor Air Quality; Putting the Issues into Perspective", American Industrial Hygiene Association, Chesapeake Section, Professional Development Conference. U.S. Naval Academy, Annapolis, MD, October 1994
1994	"Computer Applications for Managing Health, Safety and Environmental Programs" Safety Council of Maryland, June 1994
	"Emergency Response Planning" Round table American Industrial Hygiene Conference, May 1994.
	The OSHA Update Conference, Government Institutes, Inc., Washington, DC, 29-30 October 1992 — Health & Safety Audits
1992	The Environmental Management Development Summer Institute, Government Institutes, Washington, DC, 12 June 1992 — Hazard Communication Requirements — Preparing for Inspections and Working with the Regulators

Selected Speaking Engagements (cont.):

	Chairperson for "Product Risk Assessment" Roundtable, AIHA National Meeting
	Program Chairperson for "Health and Safety Auditing," Government Institute Programs
1989	Maryland Institute for Continuing Professional Education of Lawyers Advanced Real Estate Institute Environmental Issues in Land Development
1988	DMW/Cook, Howard, Downes and Tracy; Land Use Seminar Property Investigations for Hazardous Substances for Real Estate Transactions
1987	USF&G Loss Control Seminar Environmental Hazard Assessment
1986	U.S. Coast Guard Marine Safety Training School Environmental and Occupational Hazard Assessment

Professional Conference Poster Presentations:

1. Plisko, M.J. and Spencer, J.W. 1999. *Measurement for Continuous Improvement of Health, Safety, and Environmental Programs*. American Industrial Hygiene Conference and Exposition, Toronto, Canada. June.
2. Spencer, J.W. 2000. *An Example of a Quantitative/Environmental Exposure Database-An Information Resource*. American Industrial Hygiene Conference and Exposition, Orlando, Florida. May.
3. Burrelli, L., Nealley, M., Plisko, M., Spencer, J. 2004. *Exposure Assessment: An Evaluation of Benzene from the Application and Use of Spiked Penetrating Solvents*. American Industrial Hygiene Conference and Exposition, Atlanta, Georgia. May.
4. Plisko, M. and Spencer, J. 2004. *Using a Physical-Chemical Mathematical Exposure Model for estimating Occupational Exposure*. American Industrial Hygiene Conference and Exposition, Atlanta, Georgia. May.
5. Nealley, M.L., Spencer, J.W., Plisko, M.J. 2009. *The Application of Mathematical Modeling to Predict Exposures Associated with the use of an Aerosol Spray Product*. American Industrial Hygiene Conference and Exposition, Toronto, Canada. June.

Professional Publications:

1. Torrence, P.R. and Spencer, J.W. 1978. "5- 0- Alkylated Derivatives of 5-Hydrox-2[1]-deoxyuridine as Potential Antiviral Agents." *Journal of Medicinal Chemistry*. 21:228.
2. Gots, R.E., Gots, B.A., and Spencer, J. 1992. "Proving Causes of Illness in Environmental Toxicology: 'Sick Buildings' as an Example." *Fresenius Envir Bull*. 1:135.
3. Spencer, J.W. 1992. *Health and Safety Audits*. Government Institutes, Inc.
4. Rose, V.E. and Spencer, J.W. 1995. *Hazard Communication: An AIHA Protocol Guide*. AIHA Publication.
5. Spencer, J.W., Plisko, M., Balzer, R. 1999. "Asbestos Fiber Release from the Brake Pads of Overhead Industrial Cranes" *Occupational & Environmental Hygiene*. 14:397-402.
6. Nicas, M., Plisko, M.J., Spencer, J.W. 2006. "Estimating Benzene Exposure at a Solvent Parts Washer." *Journal of Occupational and Environmental Hygiene*. 3:284-291.
7. Spencer, J.W. and Plisko, M.J. 2007. "A Comparison Study Using a Mathematical Model and Actual Exposure Monitoring for Estimating Solvent Exposures During the Disassembly of Metal Parts." *Journal of Occupational and Environmental Hygiene*. 4:253-259.
8. Boelter, F.W., Spencer, J.W., Simmons, C.E. 2007. "Heavy Equipment Maintenance Exposure Assessment: Using a Time-Activity Model to Estimate Surrogate Values for Replacement of Missing Data." *Journal of Occupational and Environmental Hygiene*. 4:525-537.
9. Plisko, M.J. and Spencer, J.W. 2008. "Evaluation of a Mathematical Model for Estimating Solvent Exposure in the Workplace." *Journal of Chemical Health and Safety*. (15) 3:14-21.
10. Williams, et al. 2011. "Dermal Absorption of Benzene in Occupational Settings: Estimating Flux and Applications for Risk Assessment. *Critical Reviews in Toxicology*. 41(2):111-142.
11. Hofstetter, et al. 2012. "Evaluation of Recommended REACH Exposure Modeling Tools and Near-Field, Far-Field Model in Assessing Occupational Exposure to Toluene from Spray Paint." *Annals of Occupational Hygiene*. 57 (2): 210-220.
12. Garcia, E., Newfang, D., Coyle, J.P., Blake, C.L., Spencer, J.W., Burrelli, L.G., Johnson, G.T., Harbison, R.D. 2018. "Evaluation of Airborne Asbestos Exposure from Routine Handling of Asbestos-Containing Wire Gauze Pads in the Research Laboratory" *Regulatory Toxicology and Pharmacology*.